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PARASITOLOGY

A SUPPLEMENT TO THE

JOURNAL OF HYGIENE

EDITED BY

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AGRIPPINA BONA NOV. GEN. ET NOV. SP. REPRESENTING A NEW FAMILY OF GREGARINES.

BY C. STRICKLAND, M.A., B.C.

Assistant to the Quick Professor of Biology in the University of Cambridge.

(*From the Quick Laboratory, Cambridge.*)

(With Plate IV and 33 Text-figures.)

THE parasite forming the subject of the present paper was discovered inhabiting the alimentary tract of larvae of the common rat-flea of this country, *Ceratophyllus fasciatus* Bosc. (see Pl. IV).

The terminology employed in describing this species is that of Minchin[1], according to whom it belongs to the group of cephaline eugregarines, of which it constitutes a new family and genus. The nuclear changes in the parasite have been closely studied. These changes are very remarkable and difficult to explain on either morphological or physiological grounds, and are altogether different to the nuclear changes described by Wenyon (1911) in an allied gregarine *Lankesteria culicis*.

Nearly every flea larva was found to be infected with the parasite, whereas many *imagines* were examined with negative result. The larval excreta contained the parasite leading a free existence: a dish in which several larvae had been placed for some hours revealed many of the parasites just visible to the naked eye, while under the low power of a dissecting microscope they appeared as little pearl-like bodies in the midst of the patches of excreta. The parasite appears to exercise no injurious effect upon its host, for all the fleas in the box in which it was found seemed perfectly healthy.

[1] *Sporozoa* in Lankester's *Treatise on Zoology*, 1903.

Methods.

The parasite was studied both alive and in stained preparations. Living forms were examined by drawing out the gut of the larva in a drop of normal salt solution and eovering the preparation with a eoverslip. The gut was either left intaet or it was purposely ruptured with the objeet of liberating the organisms it contained. It is useful sometimes to substitute egg-albumin for the salt solution, as the eoverslip does not then exert so mueh pressure on the gut, besides which the structure of the nucleus of the living parasite can be made out better in those forms whieh break out of the gut.

The spores or eysts in the excreta were observed from day to day *in situ*; they were also examined in egg-albumin or salt solution. The cysts were easily picked up on the point of a fine needle, especially if they were first covered with a little egg-albumin. Useful information

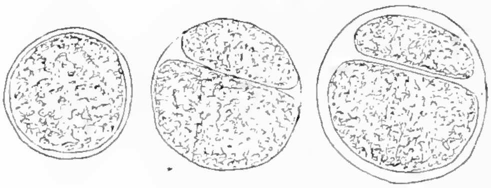


Fig. 1. Showing effect of compressing a cyst.

as to the strueture of the parasite was often gained by eompressing it, this being aeeomplished by abstraeting, by means of filter-paper, a little of the medium from beneath the coverslip (see Fig. 1). Parasites killed with a drop of osmie acid solution, run under the coverslip, were speeially favourable for the study of the myonemes.

Generally for staining purposes the parasites were liberated from the gut *in a small drop of albumin solution*, in whieh they were transferred to eorrosive-aleohol fixing fluid, being afterwards washed and stained. The cysts or spores in the exereta were treated in the same way. This method is exeellent for sueh relatively large objects, whieh eannot be fixed to a slide by drying. In staining, the best results were obtained with Heidenhain's iron-haematoxylin or Seidelin's modifieation thereof. Sometimes Flemming's fixative was used followed by the safranin-methylene-blue method of staining.

The parasites and their cysts were also studied in sections. The whole gut was plaeed in a drop of albumin on a plate of paraffin. This was put whole into the fixing fluid, and it could be then manipulated with ease throughout the washing and embedding proeesses.

The sporozoite (Fig. 2)[1], as seen free in the lumen of the midgut, is a sickle-shaped body about 4–4·5 μ in length by 1 μ in breadth. It consists of a very hyaline cytoplasm, without any partition, surrounding a nucleus. The latter is rather variable in position and appears to consist of a homogeneous oval mass of chromatin. The organism does not seem to possess any power of active movement: it doubtless enters an epithelial cell of the midgut, as is the case in related species, and grows there.

The small trophozoite (Fig. 3) is the next stage of development found in the midgut of the flea larva and it is either attached to an epithelial cell, or lies free in the gut. These free forms may only be free by virtue of the manipulations to which they are subject, but it is probable that the parasite can disengage and reattach itself at will. The small trophozoite is completely differentiated into epimerite, protomerite, and deutomerite. Its dimensions are about 12·5–16·5 × ·5 μ. In

Fig. 2.

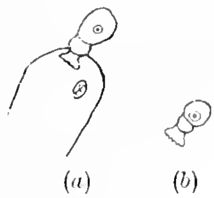


Fig. 3.

Fig. 4.

Fig. 2. Sporozoites.

Fig. 3. Young trophozoites (*a*) attached to epithelial cell, the nucleus of which is shrivelled, (*b*) free.

Fig. 4. Nucleus of small trophozoites.

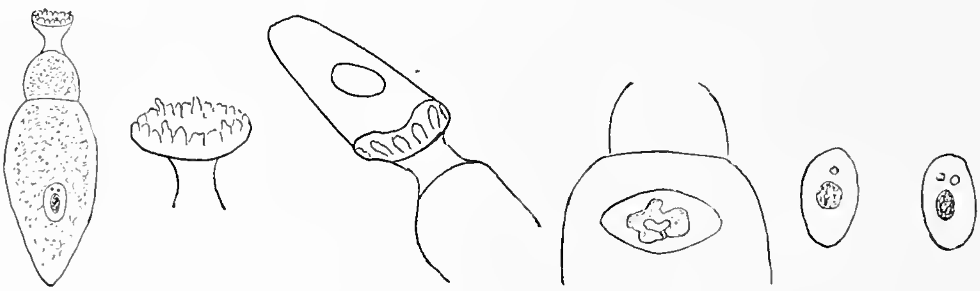
structure the cytoplasm is differentiated into ecto- and endoplasm, in the latter of which lies a small spherical nucleus (Fig. 4) limited by a nuclear membrane, and consisting of a lightly staining material containing in its centre a small sphere of chromatin formed by a close skein of this material (see Fig. 4).

The adult trophozoites (Fig. 5). All stages of the growth of the trophozoite are met with, and occur either attached or free. The arbitrarily selected type is about 55 μ long. The epimerite is symmetrical and cup-shaped with many labile digitations (Fig. 6) and is connected to the protomerite by a narrow neck. The bottom of the depression of the epimerite appears to be sometimes protruded slightly in the form of a small cone. The cytoplasm of these forms consists

[1] The figures given in the text are not drawn to the same scale; the correct dimensions are given in the text.

externally of a hyaline ectoplasm limited by a cuticle, and internally of a finely granular endoplasm.

At a variable position in the endoplasm of the deutomerite lies the now oval nucleus in which the central skein of chromatin is also oval and not so closely woven as in the nucleus of the younger forms, so that its band-like structure is more distinct. Later on this becomes completely opened out into a riband lying across the nucleus (Fig. 7), and about this time there appears in the "karyolymph," usually at the anterior end of the nucleus, one or two small spherical bodies (Figs. 5, 8), the structure and function of which I have been unable to determine. I shall merely describe what I have seen with regard to these spherical bodies. They are achromatic and of variable size and may occur singly or in pairs lying side by side. I have not been able to see any indication as to how they arise, beyond the fact that the larger of them sometimes show other similar bodies inside themselves, which is some indication that fresh ones were being formed by budding. (See Fig. 15.)



(*a*) (*b*)

Fig. 5. Fig. 6. Fig. 7. Fig. 8.

Fig. 5. Adult trophozoite.
Fig. 6. Epimerite of (*a*) free, (*b*) attached trophozoite.
Fig. 7. Chromatin band in the nucleus of a trophozoite.
Fig. 8. Nuclei of medium-sized trophozoites showing spherical "polar bodies."

The sporont (Plate IV and Fig. 9). When the adult trophozoite loses its epimerite, as it finally does, it lives free in the gut as a sporont, having an average size of $175 \times 75\,\mu$. At first the sporont is elongate like the trophozoite but eventually it swells greatly and the nucleus then passes backwards. The ectoplasm of the protomerite develops a thickened cap at its anterior extremity: the endoplasm is densely granular and appears brown to transmitted light. The granules of the endoplasm are not of the usual nature found in gregarines, as they do not consist of paraglycogen, or fat: some of them appear to be chromatic,

and are seen specially in specimens stained by safranin-methylene-blue (see Fig. 10). The myonemes (Fig. 11) are arranged in two layers, one superficial, the other deep, the former longitudinal, the latter transverse. They are very superficial structures for they may be seen in optical section to run right up to the edge of the ectoplasm. Each is distant from its neighbour by about 1·7 μ.

The changes which occur in the structure of the nucleus of the sporont are as follows. At first it consists of an oval mass containing a closely wound band staining with chromatin dyes. At each pole is a moderately large spherical body (Fig. 12) similar to those described in the trophozoite. From this form the most striking change is the opening out of the chromatin band with the formation of most variable

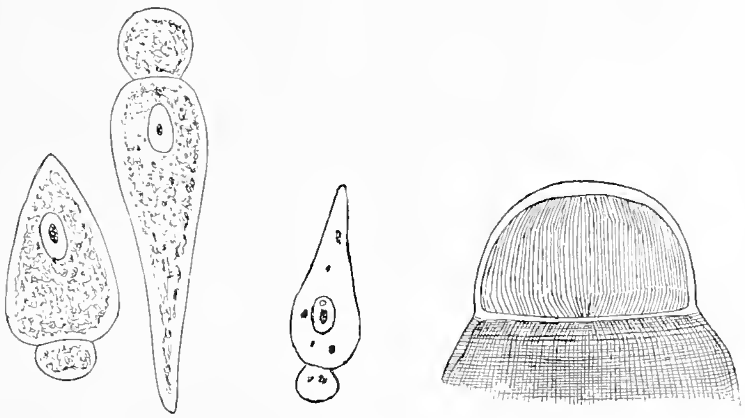


Fig. 9. Fig. 10. Fig. 11.

Fig. 9. Sporonts.
Fig. 10. Chromatic granules in the endoplasm of sporont.
Fig. 11. Anterior end of sporont highly magnified. The transverse myonemes in the protomerite are not figured. The longitudinal layer in the protomerite should have been figured as going right up to the edge of the parasite in optical section.

and fantastic figures (see Fig. 13; also Figs. 14, 15); meanwhile the chromatin band stains more and more faintly until at last it is no longer to be seen. The "polar" bodies also show striking changes. They are apparently variable in size and disposition (Fig. 14), and it is possible that they reproduce their own kind (Fig. 15). I have found no correlation between the unwinding of the chromatin skein and the presence of these bodies, but it is very striking how the main features of these changes are accompanied by conjugation of the sporonts.

The cyst. I assume that two sporonts become associated in the gut and a cyst wall is formed around them. I have never seen conjugation

take place *in vivo*, probably owing to the rapidity of the process, and I have failed to find indications of conjugation in stained preparations. Only sporonts and completed cysts could be detected. In one cyst, however (Fig. 17), there is slight evidence that the sporonts join tail to tail for the cap-like thickening of the ectoplasm in the protomerite of the sporont is seen in this cyst at opposite poles. The changes of structure which it undergoes after conjugation seem to proceed equally well whether the cyst is in the gut or in the excreta, yet as the earlier changes are usually seen in the gut and the later ones in the excreta we arbitrarily describe the following changes as exclusively occurring in these situations.

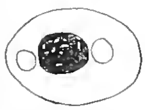


Fig. 12.

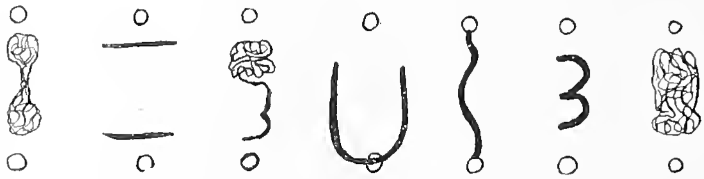


Fig. 13.

Fig. 12. Nucleus of sporont.
Fig. 13. Chromatin band of the sporont assuming different forms.

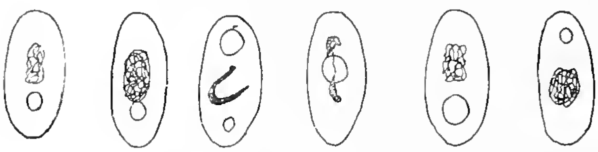


Fig. 14. Types of nuclei of the sporont later than in Fig. 12.

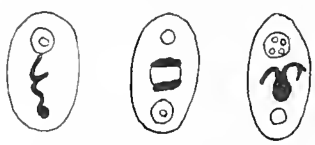


Fig. 15. Polar bodies containing others.

In the gut the cyst consists of a roughly spherical body about 85 μ in diameter when uncompressed. At first it is composed of two halves (Fig. 18) enclosed in a common ectoplasm derived from the engaged sporonts. The endoplasm is densely granular. Sometimes, especially just after association has taken place, the nucleus of each hemisphere can be seen as a clear vesicular area in the midst of the endoplasm (Figs. 16, 19), but more often nothing can be made out (Figs. 16, 18). At any rate nuclear changes proceed rapidly, and the cyst wall meanwhile becomes thicker. The partition of ectoplasm between the two halves of the cyst is finally lost, and this now consists of a

homogeneous spherical mass of granular endoplasm surrounded by the common ectoplasm. When stained the size of the cyst is reduced to about 55 μ and the following nuclear changes can be made out. At first, while the two sporonts forming the cyst are still distinct, the nucleus of each half can be readily seen, but later, when the partition has disappeared, the nucleus cannot be seen under any circumstances (cf. Figs. 20, 21). However while it can be made out, it is seen to grow enormously in size, and its ultimate disappearance may be due to its having become commensurate with the cell. Meanwhile it is invested by its very definite nuclear membrane.

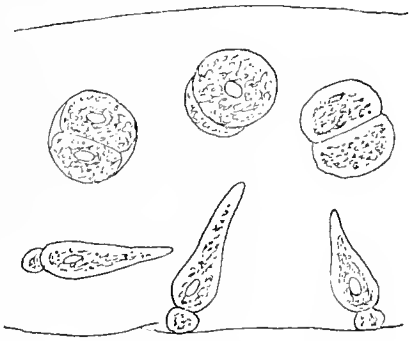


Fig. 16.

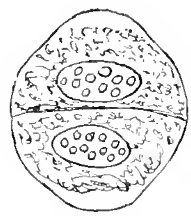


Fig. 17.

Fig. 16. Preparation of gut showing sporonts and newly formed cysts.
Fig. 17. Cyst showing indications at its poles of the anterior end of the sporonts which formed it.

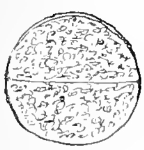


Fig. 18.

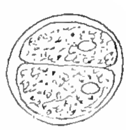


Fig. 19.

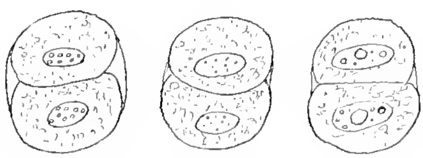


Fig. 20.

Figs. 18, 19. Cyst from the gut of a flea-larva.
Fig. 20. Nucleus in a cyst stained.

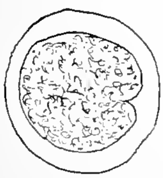


Fig. 21.

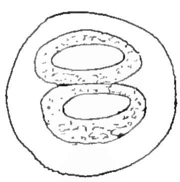


Fig. 22.

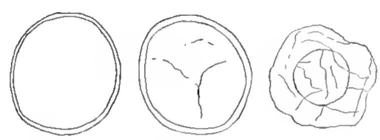


Fig. 23.

Fig. 21. Cyst stained showing fusion of the sporonts.
Fig. 22. Nucleus in a cyst stained.
Fig. 23. Cyst examined *in vivo* at stated intervals after being excreted from the larva.

The most striking change that has occurred in it is the loss of all its basic staining substance or chromatin, which cannot be seen in any form. The band-like riband of chromatin seen in the sporont has disappeared. The spherical bodies occurring in the nucleus of the sporont are now very numerous; the nucleus contains many of them irregularly scattered through the "karyolymph," and they may vary considerably in size (Fig. 20).

The significance of these changes seems very obscure. The outstanding feature is the loss of all chromatin as such: the only explanation is that it is dissolved and utilised in some way which is essential to the cell at this stage. The chromatin band of the vegetative cell is I think a kind of plastid or store-house of chromatin intended for use during the sexual processes which are now taking place. In this form the cyst is voided in the excreta.

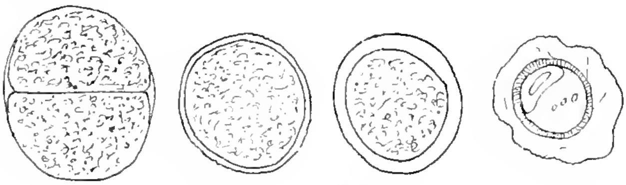
In the excreta the cyst appears to the naked eye as a minute pearly white object, but a little later (24 hours) it begins to be crinkled on the surface and on the second day is smaller and also presents a new and characteristic appearance. It consists of a well-defined spherical brown core surrounded by a crinkled fibrous-looking epicyst which is not itself spherical but rather expanded around the equator of the cyst as a sort of flange (Fig. 23, 3rd day). The size of such a cyst is about 75 μ in diameter.

At first the nucleus can sometimes be seen as a clear area in each hemisphere, and the partition between these two may be very distinct. When examined *in vivo* under the microscope (Fig. 24), during the first 24 hours there is considerable contraction of the cyst, and the ectoplasmic partition quite disappears. The epicyst increases as the endoplasmic core decreases in size. The latter appears to be merely granular up to about 54 hours after the cyst becomes free, and then suddenly appears filled with a mass of oval spores. Later on the mere wetting of a cyst filled with spores is accompanied by their discharge, their place being taken by a minute bubble of water (or air?). Two cysts, lying side by side, may differ in respect to the time they occupy in undergoing these changes, in one instance for example the third stage seen in Fig. 24 had not been passed after 72 hours.

A new structure also appears in the cyst at a variable time after, and sometimes even before, it is excreted. This is a striated band which surrounds the endoplasm but is internal to the fibrous-looking epicyst. It probably represents a system of minute tubes radially disposed round the endoplasm. It may be derived from the ectosarc of the sporont

(Figs. 24 (*d*), 26). Fig. 25 represents a highly magnified view of this striated band.

The details of the differentiation of the endoplasm into spores are very difficult to make out either in the living parasites or in stained preparations. Clear areas gradually loom up through the granular protoplasm, which is finally all used up, while the clear areas become more definite and finally resolve themselves into the highly refringent spores. When



(1 hour) (*a*) (24 hours) (*b*) (48 hours) (*c*) (54 hours) (*d*)

Fig. 24. Cyst examined *in vivo* at stated intervals after being excreted from the larva.

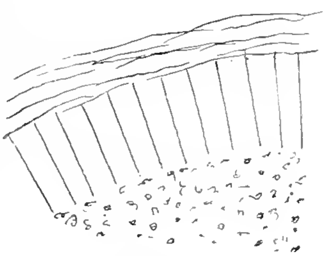


Fig. 25.

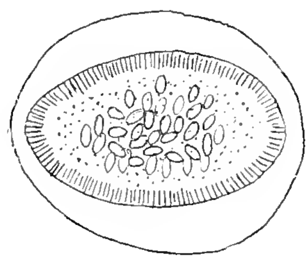


Fig. 26.

Fig. 25. The wall of the cyst highly magnified.
Fig. 26. Showing the striated endocyst and the formation of spores.

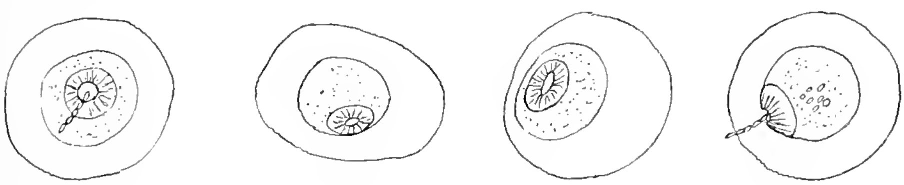


Fig. 27. Stained cysts showing method of dehiscence by rupture.

fully formed, each spore is a refringent oval body containing eight minute rings of chromatin which are the nuclei of the eight enclosed sporozoites (Fig. 26).

The spores are liberated by the simple rupture of the cyst at what seems to be a specialised area of its surface which protrudes and stains more deeply. The rupture of the cyst results in the formation of a distinct oval or circular hole (Figs. 27, 28) through which the spores

emerge in chains two or three abreast (Figs. 29, 30). These chains are excessively friable and it is impossible to pick them up without their breaking.

The spore (Fig. 31) is a uniformly oval body measuring about $7\mu \times 3\mu$. It is provided with a single covering or epispore which is highly refractile and slightly thickened at each pole. The interior is occupied by eight sporozoites closely packed together, each with a minute ring of chromatin, or nucleus.

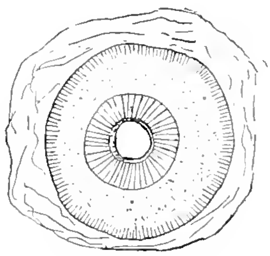


Fig. 28.

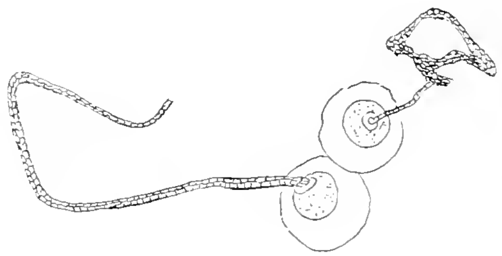


Fig. 29.

Fig. 28. Cyst showing rupture highly magnified.
Fig. 29. Cyst discharging spores.

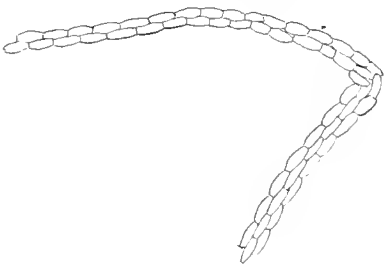


Fig. 30.

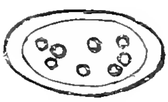


Fig. 31.

Fig. 30. A spore chain.
Fig. 31. A spore showing the rings of chromatin in the nuclei of sporozoites.

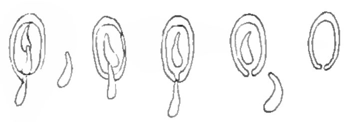


Fig. 32. Sporozoites coming out of spore.

When these spores are acted on by the gut juice of a flea-larva the eight sporozoites escape, leaving the empty spore case behind, and each of them may then undergo the same cycle of development which has been described above. I have seen the actual process taking place in the gut of a flea-larva: a spore was watched which had doubtless been

ingested by the larva whilst feeding. A sporozoite was observed coming out of one end of the spore through a small opening. Six minutes later the next sporozoite glided out with no perceptible movement, twelve minutes later the last was seen to come out after a few slight side to side movements in the spore case. The spore case was now empty. The sporozoites were watched for some time but did not enter the epithelium of the gut (Fig. 32). Fig. 33 shows the relative size and form of each stage of the life cycle.

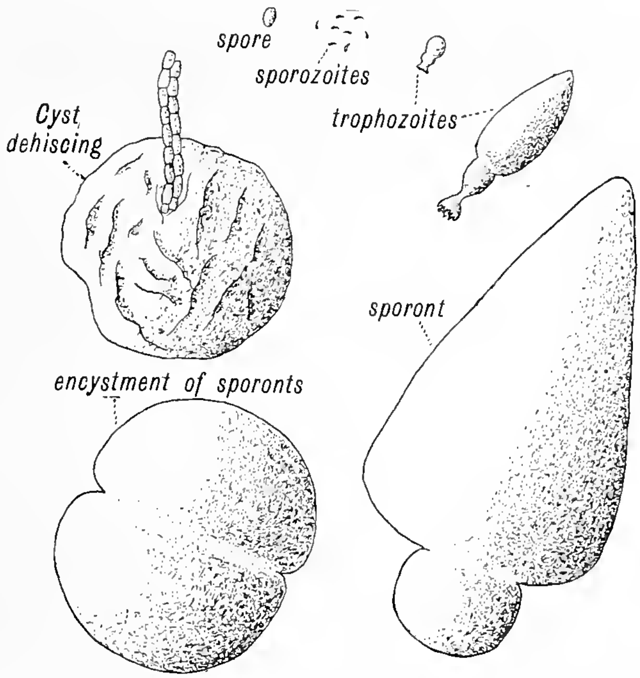


Fig. 33. Illustrating the relative sizes of the various stages in the life-history of *Agrippina bona*, each stage drawn to the same scale.

Taxonomy.

Of the families of cephaline eugregarines the parasite above described has very little resemblance to those with asymmetrical spores, viz. Menosporidae, Stylorhynchidae and Oolicystidae, or to the Acanthosporidae, which have symmetrical spores but armed with spines.

It has more resemblances to the Didymophyidae, Gregarinidae, Dactylophoridae and Actinocephalidae, all of which have symmetrical unarmed spores. Yet these families each possess well-defined characteristics which preclude us from placing the new parasite in any one of

them; the Actinocephalidae have navicular or cylindro-biconical spores and solitary sporonts; all the members of the Dactylophoridae possess an asymmetrical epimerite, long cylindrical spores, dehiscence by a lateral pseudocyst, and moreover they all inhabit myriapods; the Gregarinidae have always a simple epimerite; the Didymophyidae form peculiar syzygies in which there is no septum in the satellite.

The parasite which we have described, therefore, is sufficiently distinct to be considered the representative of a new family for which I propose the title *Agrippinidae*: which includes those cephaline eugregarines *which form symmetrical unarmed spores, oval in shape; in which there is no syzygy formation, and which have a symmetrical digitate epimerite.*

I propose the parasite be named *Agrippina bona* nov. gen. et nov. sp. I recapitulate the dimensions of the various types;—sporozoite 4–4·5 μ; small trophozoite 12·5–16·5 μ; adult trophozoite 55 μ; sporont 175 μ; cyst 85 μ; spore 6·6–7μ.

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LÉGER (1892). Tablettes zoologiques, III. 1, 2, pp. 1–182.

WENYON (1911). Oriental Sore in Bagdad together with observations on a gregarine in *Stegomyia fasciata* etc. *Parasitology*, IV. p. 273.

EXPLANATION OF PLATE IV.

The gut of the flea larva having been torn open the photograph was taken of the living mass. The nucleated epithelium is seen to the right with a small portion of a Malpighian tube below at the edge of the field. The dark central mass is undigested food, and the gregarines are seen in large numbers gathered round it as sporonts or attached to the epithelium as trophozoites.

NOTE ON "*CRITHIDIA*" *CLETI* N. SP., PARASITIC IN THE ALIMENTARY CANAL OF *CLETUS VARIUS*, DALL.

By EDWARD HINDLE, Ph.D.,
Beit Memorial Research Fellow,

and R. C. LEWIS, M.A. (Cape),
1851 *Exhibition Scholar*.

(*From the Quick Laboratory, Cambridge.*)

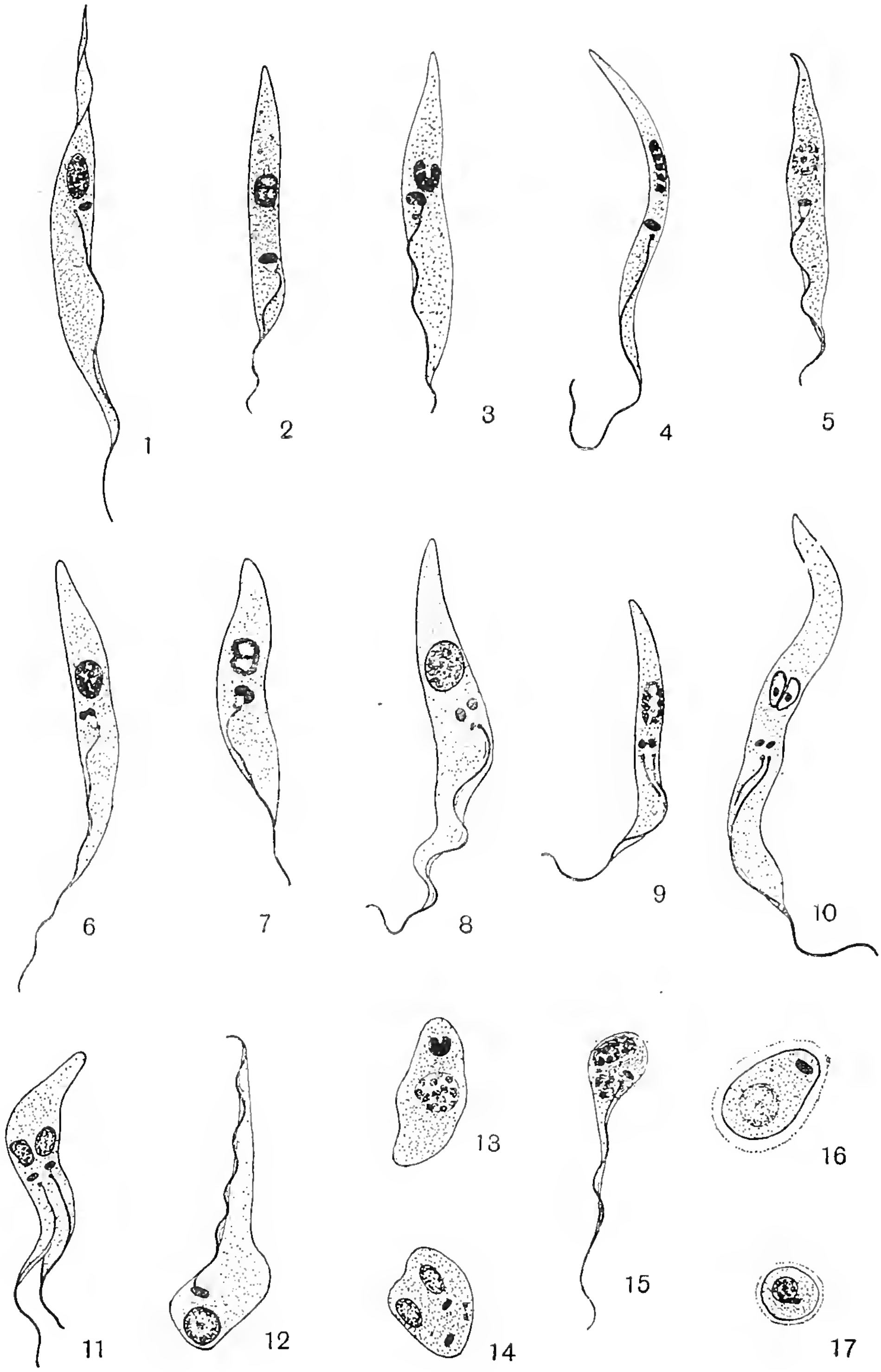
(With 17 Text-figures.)

Whilst examining the contents of a number of insects from the neighbourhood of Pretoria (Transvaal), a flagellate was found occurring in the alimentary canal of a Hemipterous insect, *Cletus varius* Dall[1]. Unfortunately, no observations were made on the living parasite and the following notes on its morphology are only derived from the study of ordinary dried smears, fixed in alcohol and stained with Giemsa. In consequence of the method of preparation, the finer nuclear details have been destroyed, but nevertheless we have been able to make out the main points in the structure of this parasite, and as no further material is available we have decided to publish our results in their present form.

Adopting Patton's (1907) terminology for the stages in its life-cycle the parasites will be described under the heading of pre-flagellate, flagellate or post-flagellate forms.

Pre-flagellate forms. Only two examples have been found which seem to represent this stage of the life-cycle. They are represented in Figs. 13 and 14 respectively. The former consists of a rather irregular ovoid body 7 μ in length and 3·5 μ in breadth. The tropho-nucleus is

[1] We are indebted to Mr Distant of the British Museum (Natural History) for the identification of this insect.



Figs. 1–17. "*Crithidia*" *cleti*. All the figures are magnified about 2500 diameters and were drawn with the aid of a camera lucida.

Fig. 1. Large flagellate form with a well developed undulating membrane. The flattened, somewhat leaf-shape of the parasite is well shown at the spirally twisted posterior extremity.
Fig. 2. Form in which the kineto-nucleus is situated rather more anteriorly than usual.
Fig. 3. Large flagellate form with dividing kineto-nucleus.
Fig. 4. Long slender form with elongated tropho-nucleus. The long flagellum arises from a conspicuous end-bead.
Fig. 5. Flagellate form showing a distinct karyosome inside the tropho-nucleus.
Figs. 6–11. Various stages in the longitudinal division of *Crithidia*.
Figs. 6, 7. Forms showing commencement of division of kineto-nucleus and end-bead.
Fig. 8. Large flagellate possessing two kineto-nuclei and dividing end-bead.
Fig. 9. Small dividing form showing the commencement of the formation of a new flagellum from the divided end-bead.
Fig. 10. Large flagellate form in which both nuclei and end-bead have divided.
Fig. 11. Commencement of fission at the anterior extremity.
Fig. 12. Form showing concentration of protoplasm and both nuclei towards the posterior extremity.
Fig. 13. Pre-flagellate stage with dividing kineto-nucleus.
Fig. 14. Pre-flagellate form in which both nuclei have divided. The end-bead is in process of division.
Fig. 15. Stage in the formation of a cyst. The protoplasm is very dense and contains granules.
Figs. 16, 17. Encysted post-flagellate forms each showing distinct tropho- and kineto-nuclei.

very large and is stained rather faintly, and the kineto-nucleus is also large and horse-shoe shaped and is very densely stained; the latter may be in process of division. Fig. 14 represents a rather interesting form which is at an advanced stage of division. Both nuclei have already divided and are situated at opposite sides of the protoplasm, and, in addition, the end-bead is clearly seen in process of division. No trace of this structure could be found in the other pre-flagellate, or in any of the cysts and therefore it seems probable that the end-bead appears shortly before the flagellum is formed and disappears in the post-flagellate stage, together with the flagellum.

Flagellate forms. This stage is extremely abundant in the films, the flagellates often occurring in large masses apparently as the result of rapid multiplication. Many of the parasites are in process of division, shown by the appearance of the kineto-nucleus or its associated end-bead. The usual variations with regard to size, shape and the relative position of the two nuclei are well shown. The tropho-nucleus is usually situated about the middle of the cytoplasm (Figs. 1, 3), but the kineto-nucleus may vary from a position immediately in front of the tropho-nucleus to one half-way between the latter and the anterior

extremity (Fig. 2). The latter form closely resembles "*Herpetomonas*" in the position of its nuclei and the absence of an undulating membrane.

A typical parasite, however, is that represented in Fig. 1. In this large flagellate form the flattened leaf-like shape of the body is extremely well shown towards the posterior extremity, which displays a slight spiral twist. The cytoplasm is quite free from any of the granules so commonly found in related species. The tropho-nucleus is situated slightly towards the posterior end. Occasionally it presents the appearance of a densely staining central karyosome surrounded by a layer of more diffusely staining chromatin (Fig. 5). More often, however, it seems to consist of numerous chromatic granules arranged either irregularly (Fig. 1) or in a linear series (Fig. 4). The kineto-nucleus is usually situated immediately in front of the tropho-nucleus and slightly to one side of the middle line. In close relation to it a small but distinct end-bead may usually be seen, situated at the apex of a small vacuole which lies immediately in front of the kineto-nucleus (Figs. 5–7). From the end-bead arises the flagellum running forward along the surface of the body, where it forms a distinct undulating membrane, until it becomes free at the anterior extremity. The dimensions of a typical flagellete form are as follows: total length from the posterior extremity to the tip of the flagellum, $25\,\mu$; breadth, $2\,\mu$; total length of flagellum, $16\,\mu$; length of free part of flagellum, $4\,\mu$.

Various stages in the division of the flagellate forms are represented in Figs. 6–11. The first sign of the process may usually be seen in the kineto-nucleus or its associated end-bead (Figs. 6–8). All the details of this division cannot be followed in the preparations before us but the kineto-nucleus seems to elongate and then constricts into two daughter nuclei (Figs. 6–8). The end-bead divides independently of the kineto-nucleus (Fig. 8) and from the new end-bead another flagellum arises (Figs. 9, 10). The new flagellum thus arises independently and is not formed by the splitting of the original one. By the time that these divisions are complete the tropho-nucleus usually shows signs of dividing, often evidenced by the arrangement of the intra-nuclear granules in two longitudinal rows one at each side of the nucleus (Fig. 9). The tropho-nucleus then divides into two equal daughter nuclei (Fig. 10) and by the time this division is complete the new flagellum is usually well developed. When the latter reaches the anterior end, the body of the parasite begins to split into two daughter parasites, this splitting always

beginning from the anterior extremity (Fig. 11), as usual in these flagellates. The two daughter parasites often remain attached for some little time before finally separating.

Post-flagellate forms. The only parasite which appears to be a stage in the formation of these post-flagellate forms is that represented in Fig. 15. In this case the protoplasm is concentrated towards the posterior extremity and at one side there is a slight thickening of the ectoplasm suggesting the formation of a cyst wall. This parasite, however, differs from its neighbours in the presence of darkly staining granules and therefore may be a degenerating form.

The fully formed cysts (Figs. 16, 17) are usually spherical bodies about 3 μ in diameter, but in one case the dimensions of 3·5 μ by 6 μ were attained. In both cases these dimensions do not include the thickness of the cyst wall.

Each cyst consists of a mass of densely staining cytoplasm containing the tropho- and kineto-nuclei; the end-bead is apparently absent. The cyst is surrounded by a cyst wall which in consequence of its non-staining properties appears as a hyaline layer. From a consideration of the life-cycle of other species of "*Crithidia*," presumably these are the forms which are excreted and serve to convey the infection to other hosts.

We are in some doubt as to what generic name to apply to this species. There is no doubt that it falls under Patton's definition of the genus *Crithidia* (1908), but as one of us has shown (Hindle, 1912), until the type species of *Leptomonas*, Kent, has been re-examined it is impossible to apply any generic name to these flagellates!

For the sake of convenience, however, we are placing it provisionally in the genus *Crithidia* (*sensu* Patton), and in view of its host, the specific name *cleti* is proposed. "*Crithidia*" *cleti* is a flagellate occurring in the intestinal tract of *Cletus varius* Dall. It is not a stage in the life-cycle of any trypanosome occurring in the blood of vertebrates, for its host feeds entirely on the juices of plants.

REFERENCES.

HINDLE, E. (1912). What is the genus *Leptomonas*, Kent? *Parasitology*, v. 128.

PATTON, W. S. (1908). The life cycle of a species of *Crithidia* parasitic in the intestinal tract of *Gerris fossarum* Fabr. *Arch. f. Protistenkunde*, XII. 131–146.

THE ANATOMY OF *STILESIA GLOBIPUNCTATA* (RIVOLTA).

By LEWIS HENRY GOUGH, Ph.D.
Entomologist, Department of Agriculture, Egypt.

(With 2 Text-figures.)

In my "Revision of the Genus *Stilesia*[1]" I had to leave several points in the anatomy of *Stilesia globipunctata* (Riv.) undecided, as the material I had of the worm was too macerated to allow of any definite conclusions. At the same time I pointed out, that the topography of the male canals as described by Stiles and Hassall[2] disagreed with the conditions obtaining in *Stilesia hepatica* Wolffhügel, in other respects a very closely related species. The necessity for a re-examination of *Stilesia globipunctata* (Riv.) in order to decide whether the differences really exist, or whether they were supposed to do so, on account of the material examined by Stiles being rather macerated (*loc. cit.* p. 74), was all the greater as *Stilesia globipunctata* (Riv.) is the type species of the genus *Stilesia*. Through the kindness of Mr Littlewood, Chief Inspector of the Veterinary Department, I was able to obtain living examples of *Stilesia globipunctata* (Riv.) at the Cairo abattoir on Jan. 27, 1912. These worms have been fixed with Zenker's solution, hardened and cut.

Stilesia globipunctata (Riv.) when living very closely resembles *Stilesia hepatica* Wolffhügel. Both worms have in common a certain gelatinous appearance commented on by Dr Giles, quoted by Stiles and Hassall (*loc. cit.* p. 74). Both are of approximately the same size, and are extremely contractile. My new specimens, when found,

[1] *Quart. Journ. Micr. Sci.* Feb. 1911.

[2] Stiles, C. W. and Hassall, A. (1893). A Revision of the Adult Cestodes of cattle, sheep, and allied animals. *U. S. Dept. Agric., Bur. Animal Industry*, Bulletin No. 4.

were contracted to their fullest extent, the strobila being spirally twisted, or frilled by the extremity of the contraction. Some of the specimens, however, could be relaxed sufficiently before fixing, to enable good sections to be made.

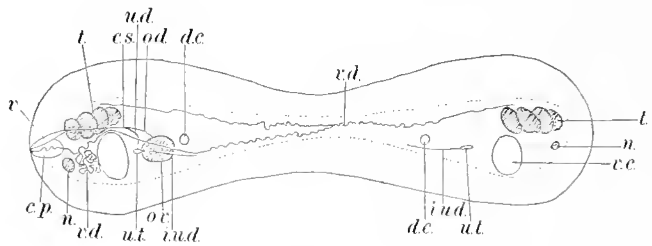
In my specimens the scolex is generally rather smaller than the size given by Stiles, being about 0·5 mm. in diameter instead of 0·768–0·9 mm. The strobila directly behind the scolex is only one-half to two-thirds this width; it begins to widen 2–3 mms. behind the head, and attains 1·3 mm. at 1 cm. from the scolex. The widest segments are about 2·5 mm. wide, by 0·07 mm. long, and the thickness varies from 0·25 mm., at the level of the ventral canals, to 0·1 mm. along the median line. In consequence of the thinness of the median portion of the segments, the strobila has a tendency to fold over on itself longitudinally.

There are four to eight testes on either side of the segment, six being a common number. These all lie laterally or dorsally to the ventral canal, and are arranged in one row, or in two consecutive transverse rows of three or four, according to the state of contraction of the worm. (Text-figure 1 *t.*) In *Stilesia hepatica* Wolff. some of the testes are median to the ventral canal. (Text-figure 2 *t.*) The vas deferens of each side starts dorsally to the testes, as in *Stilesia hepatica* Wolff. (*v.d.*) and crosses above both the ventral canal and the dorsal canal; the right and left branches of the vas deferens meet in the median field, here they combine and form a common vas deferens which runs across the segment towards the pore side, crossing the dorsal canal ventrally, and the ventral canal dorsally, just as in *Stilesia hepatica* Wolff. (see figures.) Arrived lateral to the ventral canal the vas deferens becomes swollen with spermatozoa, and forms many closely wound convolutions, which lie either ventral to the testes, or when the worm is well expanded anterior to them. The absence of convolutions of the vas deferens can consequently no longer be used to differentiate *Stilesia globipunctata* (Riv.) from *Stilesia vittata* Railliet. The cirrus pouch in my sections is invariably ventral to the vagina. It lies in the anterior corner of the segment, alternating irregularly on the right or left of the strobila. When the strobila is very contracted, the cirrus pouch and vagina are often extruded together so as to form a small knob-like projection covered of course by subcuticula and cuticula.

The ovary (*ov.*) lies median to the ventral canal and lateral to the dorsal canal of the pore side. The oviduct (*o.d.*) meets the seminal

canal (*c.s.*) and the uterine duct (*u.d.*) and joins them dorsally and median to the ventral canal. The seminal canal runs straight from the point of its junction with the oviduct to the vagina (*v.*), which, as already stated, appears to be always dorsal to the cirrus pouch. The uterine duct, after branching off from the canalis seminalis and oviduct, turns ventrally and connects with the developing uterus (*ut.*), which,

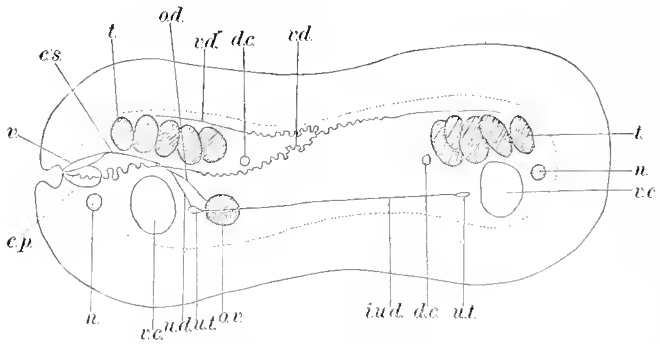
Dorsal Surface



Ventral Surface

Fig. 1. Diagrammatic transverse section of *Stilesia globipunctata* (Rivolta).

Dorsal Surface



Ventral Surface

Fig. 2. Diagrammatic transverse section of *Stilesia hepatica* Wolffhügel.

List of abbreviations: *v.c.* ventral canal; *d.c.* dorsal canal; *n.* nerve; *t.* testes; *v.d.* vas deferens; *c.p.* cirrus pouch; *ov.* ovarium; *ut.* uterus; *o.d.* oviduct; *u.d.* uterine duct; *i.u.d.* inter-uterine duct; *c.s.* seminal canal; *v.* vagina.

as it comes into function, lies lateral to the ovary. The ovary disappears very soon after the uterus has commenced to function, but the oviduct persisting for some time marks the place where the ovary used to be. An inter-uterine duct apparently functions for a short time only (*i.u.d.*), the portions nearest to the uterus being most readily made out. The paruterine organs appear to develop in this

portion of the inter-uterine duct. The eggs come to lie in pockets on the lateral side of the paruterine organ.

The ventral canal is always well developed, often occupying half of the dorsoventral width of the medullary layer. The ventral canals of both sides are connected by a network of transverse canals, as in *Stilesia hepatica* Wolffhügel. The dorsal canals are less developed, and without transverse connections; glandular cells, such as I described for *Avitellina centripunctata* (Riv.), surround the dorsal canal.

The nerve lies ventral to the genital canals on the pore side, ventral to the testes on the non-pore side.

The great similarity in the general arrangement of *Stilesia globipunctata* (Riv.) and of *Stilesia hepatica* Wolff. is very easily seen by comparing the two diagrams.

Stilesia hepatica Wolff. does not appear to be known in Egypt; *Stilesia globipunctata* (Riv.) was certainly never found in any of the many sheep I examined in the Transvaal.

My thanks are due to Prof. W. Garstang of Leeds University, for having kindly revised all the proofs of my paper on the "Revision of the Genus *Stilesia*," which appeared when I was in Trinidad. This is the first opportunity I have had of thanking him publicly for the immense amount of trouble it must have involved.

THIRD LIST OF GENERIC NAMES FOR THE "OFFICIAL LIST OF ZOOLOGICAL NAMES."

Communicated by C. W. STILES, Ph.D.
Secretary International Commission on Zoological Nomenclature.

9[1]. The following generic names of animals reported as parasites of man have been submitted to the International Commission on Zoological Nomenclature, by the Helminthological Society of Washington, for *inclusion* in the "Official List of Zoological Names":

Cestoda:

Davainea R. Blanchard and Railliet, in R. Bl., 1891 *t*, 428–440, type *proglottina* (in chickens; France).

Diplogonoporus Loennberg, 1892 *a*, 4–16, type *balaenopterae* (in *Balaenoptera borealis*; Finmarken).

Dipylidium Leuckart, 1863 *a*, 400, type *caninum* (in dogs; Europe).

Echinococcus Rudolphi, 1801 *a*, 52–53, 55, type *granulosus* (in sheep; Europe).

Taenia Linnaeus, 1758 *a*, 819–820, type *solium* (in *Homo*; Europe).

Nematoda:

Ancylostoma[2] [Dubini, 1843 *a*, 5–13] emendation Creplin, 1845 *a*, 325, type *duodenale* (in *Homo*; Italy).

Ascaris Linnaeus, 1758 *a*, 644, 648, type *lumbricoides* (in *Homo*; Europe).

Dracunculus "Kniphof, 1759, 12" [not verified]; Gallandat, 1773 *a*, 103–116, type *medinensis* (in *Homo*).

Gnathostoma Owen, 1836 *f*, 123–126, type *spinigerum* (in *Felis tigris*; London).

Necator Stiles, 1903 *y*, 312, type *americanus* (in *Homo*; U.S.A.).

Strongyloides Grassi, 1879 *f*, 497, type *intestinalis*=*stercoralis* (in *Homo*).

Trichostrongylus Looss, 1905 *o*, 413–417, type *retortaeformis* (in *Lepus timidus*; Europe).

Gordiacea:

Gordius Linnaeus, 1758 *a*, 644, 647, type *aquaticus* (free; Europe).

Paragordius Camerano, 1897 *g*, 368, 399–402, type *varius* (free; U.S.A.).

[1] Paragraphs are numbered continuously with the earlier lists.

[2] See Art. 19, and Opinions 26, 27, 34, and 36.

Acanthocephala :

Gigantorhynchus Hamann, 1892 *d*, 196, type *echinodiscus* (in *Myrmecophaga jubata*, *M. bivittata* ; Brazil).

10. The Secretary presents the following generic names for definite *rejection* from the "Official List," on the ground that they are preoccupied (see Art. 34).

Trematoda :

Acanthocephala Dies., 1858, not Laporte, 1832.
Acrodactyla Staff., 1904, not Hal., ante 1846.
Anadasmus Looss, 1899, not Walsingham, 1897.
Anisogaster Looss, 1901, not Deyr, 1863.
Astia Looss, 1899, not Koch, 1879.
Baris Looss, 1899, not Germ., 1817.
Brachymetra Stoss., 1904, not Mayr, 1865.
Creadium Looss, 1899, not Vieill., 1816.
Crossodera Duj., 1845, not Gould, 1837.
Eurycoelum Brock, 1886, not Chaudoir, 1848.
Eurysoma Duj., 1845, not Gistl., 1829.
Leioderma Staff., 1904, not Will.-Suhm, 1873.
Leptalea Looss, 1899, not Klug, 1839.
Leptosoma Staff., not Leach, 1819.
Levinsenia Stoss., 1899, not Mesnil, 1897.
Macraspis Olss., 1868 or 1869, not McL., ante 1835.
Megacetes Looss, 1899, not Thomas, 1859.
Microscapha Looss, 1899, not LeConte, 1866.
Polyorchis Stoss., 1892, not Agassiz, 1862.
Polysarcus Looss, 1899, not Fieb., 1853.
Spathidium Looss, 1899, not Duj., 1841.
Stomylus Looss, 1899, not Fahraeus, 1871.

Nematoda[1] :

Acanthophorus Linst., 1876, not Serv., 1832
Acanthosoma Mayer, 1844, not Curt., 1824.
Aspidocephalus Dies., 1851, not Motsch, 1839.
Brachynema Cobb, 1893, not Fieb., 1861.
Cephalacanthus Dies., 1853, not Lac, 1802.
Cephalonema Cobb, 1893, not Stimps, ante 1882.
Chaetosoma Claparède, 1863, not Westwood, 1851.
Cheiracanthus Dies., 1838, not Agassiz, 1833.
Cochlus Zed., 1803, not Humph., 1797.
Conocephalus Dies., 1861, not Thunb., 1812.
Cystocephalus Rail., 1895, not Leger, 1892.

[1] This list contains a few names of organisms which are not Nematoda, but which have been classified as such at one time or another.

Diceras Rud., 1810, not Lam., 1805.
Dipeltis Cobb, 1891, not Pack., 1885.
Discophora Vill., 1875, not Boisd., 1836.
Eucamptus Duj., 1845, not Chevr., 1833.
Eurystoma Marion, 1870, not Raf., 1818.
Fimbria Cobb., 1894, not Bohadsch, 1761.
Hoplocephalus Linst., 1898, not Cuv., 1829.
Leptoderes Duj., 1845, not Serv., 1839.
Litosoma Ben., 1873, not Douglas and Scott, 1865.
Mitrephorus Linst., 1877, not Schoenherr, 1837.
Oxysoma Schneid., 1866, not Gerv., 1849.
Oxystoma Buetschli, 1874, not Dum., 1806.
Oxyurus Lam., 1816, not Raf., 1810.
Paradoxites Lindem., 1865, not Goldf., 1843.
Pelodytes Schneid., 1860, not Fitz., ante 1846.
Pterocephalus Linst., 1899, not Schneid., 1887.
Ptychocephalus Dies., 1861, not Agassiz, 1843.
Rhabdogaster Metschnikoff, 1867, not Loew., 1858.
Rhabdonema Leuck., 1883, not Kuetzing, 1844.
Rhabdonema Perr., 1886, not Kuetzing, 1844.
Rhytis Mayer, 1835, not Zed., 1803.
Spilophora Bast., 1865, not Bohem., 1850.
Spinifer Linst., 1901, not Raf., 1831.
Spira Bast., 1865, not Brown, 1838.
Spirura Dies., 1861, not E. Bl., 1849.
Trichina Owen, 1835, not Meig., 1830.
Trichoderma Greef, 1869, not Steph., 1835.
Trichodes Linst., 1874, not Herbst, 1792.
Triodontus Looss, 1900, not Westwood, 1845.
Tropidurus Wiegm., 1835, not Neuwied, 1824.
Tropisurus Dies., 1835, not Neuwied, 1824.

GORDIACEA :

Paragordius Montgomery, 1898, =Camerano, 1897.

ACANTHOCEPHALA :

Arhynchus Shipley, 1896, not Dejean, 1834.
Neorhynchus Ham., 1892, not Sclater, 1869.

11. The names in question are published for the information of all persons interested. Objection to the proposed action should be filed with the Secretary (C. W. Stiles, Washington, D.C.) not later than January 1, 1913, together with ground upon which objection is based.

12. The above names will be forwarded immediately to the International Commission on Medical Zoology, and to the special sub-committees in the groups in question for special report.

13. The list will be forwarded about July 1, 1912, to the International Commission on Zoological Nomenclature, and the Secretary expects to call for a vote on these names at the next regular meeting of the Commission, in the summer of 1913.

14. The Secretary takes this opportunity to state that his policy is to bring into the list a number of names upon the adoption of which no difference of opinion seems to exist, and to reject a large number of preoccupied names, before he submits for study the names upon which differences of opinion are expressed by authors.

THE LIFE-CYCLE OF *SPIROCHAETA GALLINARUM.*

AN APPRECIATION AND A CRITICISM OF DR E. HINDLE'S RECENT PAPER.

By ANDREW BALFOUR, M.D., B.Sc., F.R.C.P., D.P.H.
Director, Wellcome Tropical Research Laboratories, Gordon College, Khartoum.

I HAVE been following the valuable contributions which Hindle has recently been making to the literature of spirochaetosis and I have been specially interested in his paper on the above subject which appeared in *Parasitology*, vol. IV. p. 463. I wish to make a few comments upon some of his findings and conclusions.

The peculiar form of transverse division which he describes probably explains why there have been such conflicting views as regards the division of this species of spirochaete. While I cannot say that I have ever actually witnessed transverse division in the case of the Sudan fowl spirochaete I have certainly seen forms such as he represents in Fig. 1, p. 464. Indeed I have illustrated several of these forms both in the Third and Fourth Reports of these laboratories.

I am particularly pleased to find that he has been able to confirm my observations on granule shedding because, as I have frequently remarked, I believe this phenomenon to be of very considerable significance and to be present in other than spirochaetal conditions. His name "coccoid bodies" for these granules seems useful but surely he is in error when he says,

"The fact that these coccoid forms are produced in large numbers after drug treatment, or when the parasites are in unfavourable conditions, has caused them to be regarded previously as merely the result of granular disintegration."

So long ago as 1907 Breinl observed *Sp. duttoni* breaking up into small red granules and stated that it was out of these that the new

generation of spirochaetes was evolved. He did not, however, show how such evolution occurred. Moreover in my later work, which apparently Hindle has not seen, for the last of my papers which he quotes is one that appeared in April 1911, I clearly expressed my belief that these granules are of an infective nature and are intimately concerned with the life-cycle of the spirochaete. I see that, like myself, Hindle has not derived very much information from preparations stained by the more ordinary methods. I trust, however, that he will find the process of vital staining of great service. I have recently sent a communication on this subject to the *British Medical Journal.* Undoubtedly the most interesting portion of Hindle's paper is that dealing with the life-cycle in the tick, *Argas persicus.* Here again it would seem that he has not had his attention drawn to my later work on this subject, especially to the paper which I read last July, before the tropical medicine section of the British Medical Association when I partly traced the very development Hindle has now fully outlined and when I exhibited specimens of tick tissues showing the changes from granule or coccoid body to bacillary and spirochaetal forms. This paper was published in the *Journal of Tropical Medicine and Hygiene* for September 1st, 1911, and later in the *British Medical Journal* for November 11th, 1911. A much earlier paper (Oct. 1st, 1909) in the former journal has also apparently been overlooked. It signalises the discovery of granules in the tick and records successful inoculation experiments with them.

The subject is further partly dealt with and very fully illustrated in the Fourth Report of these laboratories, Volume A which appeared in November 1911. The experiments there detailed were mostly carried out early in that year. I have no wish to dispute the question of priority in this portion of the research, for Hindle and I would seem latterly to have been working at the subject more or less contemporaneously and, in any case, it was Leishman who led the way. It is only fair, however, to direct attention to papers which are the outcome of several years of a research that had to be sandwiched in between administrative and routine work whenever time permitted. I see that Hindle has outstripped me for he has carefully worked out the complete cycle which I have not had time to do. There are, however, apparently differences between the development of his strains and that with which I have been working, for whereas he finds that the spirochaetes themselves penetrate the cells in the tick and there break up into coccoid bodies it would seem that in the case of the Sudan fowl spirochaete only the granules do so. It is this, along with certain other features,

which have led me to regard it as a distinct species, a finding to which I see Galli-Valerio (1911) objects and which I admit may yet be shown to be unjustified. I might have been able to say more about the cycle in the tick, for like Hindle, I have been studying the development in the eggs, but just as the results were becoming very interesting I had unfortunately to go to Cairo for Pasteur treatment and so lost a good six weeks.

In other respects Hindle's results closely approximate to mine though some of his inoculation experiments do not appear to agree with what I have found in Khartoum.

I turn finally to his remarks regarding my intra-corpuscular bodies, the condition I have recently termed the "granule phase" and which he discusses on p. 470. He admits that he has little personal knowledge of these curious inclusions and puts forward a perfectly legitimate hypothesis that they are the results of nuclear degeneration. One of his arguments in favour of this view is worded as follows:

"It is important to note that Balfour's figures show that these bodies have exactly the same staining reaction as the nucleus of the red cell, and, moreover, are identical in appearance with the products of undoubted nuclear degeneration seen in normal fowls."

If he had seen my paper in our Fourth Report before penning his article he would have found that this argument no longer holds. It is true, as might be expected, when Romanowsky staining is employed but with other stains it is not the case (vide Pl. III. fig. 8).

I long ago considered this question of nuclear degeneration. That it occurs I admit, for I have frequently seen extrusions of nuclear substance into the cytoplasm of the red cells. That my inclusions have anything to do with it I cannot believe and I think it is a pity that Hindle in his final paragraph on the subject (p. 469) should have changed hypothesis to fact and stated definitely that

"This nuclear degeneration seems to be a special feature of the fowl spirochaetosis occurring in the Sudan and in South Africa (Jowett), for it only rarely occurs in the blood of fowls infected with the strains of this disease from Brazil and Algeria respectively. With these latter strains of spirochaetosis the infected bird usually dies at the height of infection, or else recovers, and the infection rarely assumes the chronic form, necessary for the production of abundant nuclear degeneration of the red cells."

It is unfortunate that he did not see my later work for there I point out that it is the granule and not the spirochaete which enters the red

cell to form the inclusions and I have been able, I think correctly, to trace a development of the granule in the red cell, a development which results either in a discharge of an encapsuled body containing granules or of small granules themselves into the *liquor sanguinis*. The latter process strongly suggests schizogony[1]. I see that Galli-Valerio is now of opinion that the inclusions are spirochaetal in origin though he imagines that the spirochaete sheds granules or coccoid bodies when lying in the substance of the cytoplasm. So far I have no proof that this occurs. As a matter of fact the method of vital staining has, I believe, made clear much that was hitherto obscure and will enable one to make absolutely certain as to the true nature of this "granule phase." Like Hindle I have seen and reported intra-corpuscular bodies in normal birds. Their presence is difficult to explain but, as I have suggested, they may indicate an hereditary infection of the bird through the egg. It is also possible that in some cases they may merely be the remnants of an old infection while, of course, nuclear extrusions may sometimes simulate the forms I have described. Doubtless at first I may have confused two separate conditions but, while I admit the last word has still not been said on this difficult subject, I am persuaded that the true "granule phase" has nothing to do with nuclear degeneration but is as much a part of the life-cycle of the fowl spirochaete as are the developmental stages which Hindle has so carefully traced in the cells and fluids of *Argas persicus*, a piece of work which is in accord with my own findings and effectually, I think, disposes of the arguments recently advanced by Blanc (1911). The latter I believe to be mistaken owing to his unfamiliarity with the true form of the infective granule or coccoid body. Apart from anything else I would like to know how, on the nuclear degeneration hypothesis, Hindle can explain certain observations recorded in my paper in our Fourth Report (pp. 86 and 95) whereby I have shown that after inoculation of spirochaetal blood or infected tick tissue (*A. persicus*) into perfectly healthy chicks the inclusions may first appear even as early as the day following the inoculation, to be followed later, in some instances, by the appearance of spirochaetes. In such cases, and the work with *O. savignyi* p. 96 is equally suggestive, what was there to cause nuclear degeneration?

[1] I may say that the little schizonts are beautifully shown by vital staining with toluidin-blue. This specially applies to the "spore" or morula stage where the tiny granules stain deeply and are shown to be arranged in a regular and definite manner. A glance at such a preparation is sufficient to put any question of nuclear degeneration wholly out of account.

There had been no prolonged illness, nothing to affect the red cells profoundly and yet, as I say, the typical "granule phase" developed. This seems a conclusive argument against the views of Hindle and those who share his opinion on this matter, while I think a study of my later experimental work and the employment of vital staining is likely to convince those who still think the intra-corpuscular "granule phase" is only indirectly associated with the spirochaetal infection.

I am continuing the work as opportunity permits and hope ere long to publish a final paper giving a detailed account, both of the complete cycle in the chick and that in the tick which serves as the intermediary host of the fowl spirochaete.

Fantham's latest paper (1911) has just come to hand and is full of interesting matter. I note that he does not wholly agree with Hindle's views as regards transverse division, that he confirms the phenomenon of granule-shedding and that, in certain respects, his account of the life-cycle in the tick is more like my own than that given by Hindle. He also agrees with me that the intra-corpuscular granules are of spirochaetal origin although he is not inclined to attribute much importance to such granules in the bird's blood, believing that their appearance is merely a kind of anticipation of what occurs in the insect host. With this, as I have stated, I cannot agree and I do not think there are any analogies to support such a contention. Nature is not wasteful and has usually some definite reason for the occurrence of phenomena of this nature. The difficulty is to determine it. In the case of the Sudan spirochaete I think I can claim to have done so.

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NOTE ON THE FOREGOING COMMUNICATION BY DR ANDREW BALFOUR.

By E. HINDLE, Ph.D.

I am extremely sorry that Dr Balfour should think that I have overlooked his important papers on the subject of the development of *S. gallinarum*; I can only say that this was not the case. At the head of my paper, I distinctly stated that it was only a Preliminary Note, and as such I avoided any discussion of the subject from an historical point of view. Had I done so it would have been necessary to refer to the work of Marchoux, Dutton and Todd, Markham Carter, and other investigators who have advanced our knowledge of the morphology of blood spirochaetes. In the paper under discussion I merely wished to state some of the conclusions I had come to on this subject as a result of my own researches and the complete account was reserved until I had finished further work on the nature of this parasite and its life-cycle; in the future paper full reference will be given to all previous publications.

Through the kindness of Dr Balfour, whom I should like to take the opportunity of thanking for his courtesy, I have been enabled to examine some slides of the "intra-corpuscular bodies" described by him and considered as a stage in the life-cycle of *S. gallinarum*. I find that I was mistaken in supposing them to be the result of nuclear degeneration of the red cells, for they differ from any of the appearances presented by the blood of normal fowls. When I wrote my Preliminary Note, the Fourth Report of the Wellcome Research Laboratories had not yet been published and therefore I had not had the opportunity of seeing Dr Balfour's later work on the subject, in which he clearly distinguishes these bodies from any form of nuclear disintegration.

Their true nature, however, is still uncertain, in spite of Dr Balfour's very interesting results, both published and unpublished. A distinct cycle of development—schizogony, escape of schizonts, penetration of another corpuscle, etc.—seems to occur in the intra-corpuscular bodies and up to the present they have not been shown to develop either from, or into, spirochaetes. I have never seen these bodies in the blood of fowls infected with the Brazilian strain of spirochaetosis, in spite of daily examinations of numerous birds extending over some months. The bodies are so very conspicuous that their presence could not be overlooked. I have not had time to decide the question, but the possibility of a mixed infection occurring in Africa does not appear to me to have been as yet excluded.

WHAT IS THE GENUS *LEPTOMONAS* KENT?

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The nomenclature of the parasitic flagellates of invertebrates is in such a state of chaos at the present time, that I have collected the history of the three more important genera *Leptomonas*, *Herpetomonas*, and *Crithidia*, in the hope that it will end the controversies which surround the application of any one of these names.

The first of these flagellates to be described was *Herpetomonas muscae-domesticae*. In the year 1851, Burnett (1851) published a short note on the result of his investigations concerning the structure and nature of the genus *Bodo*. In this article there is a brief mention of the fact that flagellates were found parasitic in the alimentary canal of the common house-fly, and Burnett proposed for these parasites the name *Bodo muscae-domesticae*.

Under the name *Bodo muscarum*, Leidy (1856) briefly described a flagellate which he found frequently occurring in the intestine of *Musca domestica*, often in immense quantities.

Stein (1878) united *Bodo muscae-domesticae* Burnett and *B. muscarum* Leidy, but placed the parasite in the genus *Cercomonas* and under the title of *Cercomonas muscae-domesticae* this flagellate is described and figured (Pl. I, pt. 2, figs. 1–4). According to Stein's description this parasite possesses a remarkable degree of flexibility, and therefore it is a little doubtful whether it can be identified with the *Herpetomonas muscae-domesticae* of Prowazek (1904).

Finally Kent (1881) established a new genus, *Herpetomonas*, for the reception of this form. The rat trypanosome (*T. lewisi*) was also provisionally included, but the parasite of the house-fly was designated as the type of the genus.

Kent's (1881) diagnosis of the genus is as follows:

"Animalcules free-swimming, elongate or vermicular, highly flexible; the posterior extremity often the most attenuate, but not constituting a distinct caudal appendage; flagellum single, terminal; contractile vesicle usually conspicuous. Habits mostly endoparasitic" (Vol. I, p. 245).

However, two pages before this (*loc. cit.*, p. 243), Kent had already given the diagnosis of another new genus, *Leptomonas*, formed for the reception of a flagellate parasite occurring in the intestine of *Trilobus gracilis*, and which had been described and figured five years previously by Bütschli (1876).

The diagnosis of the genus *Leptomonas* was given as follows:

"Animalcules free-swimming, persistent in shape, elongate fusiform or aciculate, bearing a single long undulating flagellum at the anterior extremity; no distinct oral aperture yet detected."

The only species referred to the genus was *L. bütschlii* Kent.

If the respective diagnoses of the genera *Herpetomonas* and *Leptomonas* are compared it will be seen that the differences between them are extremely difficult to recognize at the present time. The presence of a contractile vacuole and the extreme flexibility of the body in *Herpetomonas* constitute the only distinct differences between the two, and neither of these characters is recognized in any modern definition of the genus. The contractile vacuole is not present in *Herpetomonas muscae-domesticae*, nor in any other member of the group, and yet this constitutes the main point of distinction between the above two genera. As for the flexibility of the body, this is a very uncertain character and is insufficient to distinguish two genera.

On these grounds, therefore, *Leptomonas* Kent and *Herpetomonas* Kent should be united and become one genus.

This fact was recognized by Bütschli (1884) but instead of retaining the name *Leptomonas* that of *Herpetomonas* was selected, and the former became a synonym. According to the "Law of priority" in the *International Code of Zoological Nomenclature* the genus *Leptomonas*, being described two pages before *Herpetomonas*, has priority and therefore should have been retained.

Still further confusion was caused by Senn (1900) who removed *Herpetomonas muscae-domesticae* to the genus *Leptomonas*.

The latter therefore was represented by two species, *muscae-domesticae* and *bütschlii*. The genus *Herpetomonas* was retained for the reception of the rat trypanosome, described by Kent (1881) as *Herpetomonas*

lewisi, and the newly discovered *Trypanosoma brucei* Plimmer and Bradford also was referred to it. Laveran and Mesnil showed that these two species really belong to the genus *Trypanosoma*, and accordingly Senn (1902) two years later returned to Bütschli's conception of the genus *Herpetomonas*. In the meantime yet another generic name, *Herpetosoma* Doflein (1901), had appeared, and this was included by Senn as one of the synonyms of *Herpetomonas*.

The same year Léger (1902) formed a new genus *Crithidia* for the reception of a parasite of the intestinal tract of *Anopheles maculipennis*. Although Léger's original description is very incomplete, yet the remark that its characters were intermediate between *Herpetomonas* and *Trypanosoma*, and the accompanying figures, show that he was describing a form very similar to the *Crithidia* subsequently described by Patton, Porter, Swingle and other writers, and Patton (1908) emended the original diagnosis of the genus.

At first Léger was in some doubt about the parasite, for he writes—"En raison du mode d'alimentation des *Anopheles* et de l'analogie que présentent les formes affilées de ce flagellé avec les Trypanosomes, on peut se demander si les *Crithidia* ne représenteraient pas un certain stade évolutif de quelque hématozoaire flagellé des Vertébrés" (Léger, 1902 *a*, p. 356).

In a subsequent paper, however, he (Léger, 1903) seemed convinced of the validity of the genus *Crithidia* and described two new species, *minuta* and *campanulata*. The former of these species was later removed by Léger (1904) to the genus *Herpetomonas*, and the presence of a rudimentary undulating membrane was mentioned as occurring in the latter. The shape of the body was then considered as being the only distinct character of the genus *Crithidia*, for Léger remarks that the only two species are characterised by "la forme massive, piriforme ou campanulée de leur corps" (1904, p. 616).

Prowazek (1904) in describing the life history of *Herpetomonas muscae-domesticae* gave a very complicated account of both its structure and life cycle. In this paper the parasite was described as possessing two flagella united by a membrane and arising from an anteriorly situated diplosome. It is very difficult to accept all of Prowazek's hypotheses regarding the structure of this parasite, and up to the present they have not been entirely confirmed (*v. infra*).

However, it is certain, as Roubaud (1909) first pointed out, that if *Herpetomonas muscae-domesticae* is biflagellate it differs in this character from many other species which have been referred to this genus.

In consequence Roubaud (1909), Chatton (1909) and later Dunkerly (1911) prefer to retain the genus *Herpetomonas* for the biflagellate *H. muscae-domesticae* and employ the older generic name *Leptomonas* for the uniflagellate forms.

On the other hand a number of authors (*e.g.* Patton, 1909; Mackinnon, 1910) have denied the existence of a true biflagellate stage in *H. muscae-domesticae*, explaining the appearance described by Prowazek as merely a stage in the division of the parasite. Personally, I am of the opinion that the structure and mode of division of closely related parasites constitute strong arguments against Prowazek's view, but nevertheless it is perhaps advisable to retain the genus *Herpetomonas* for *H. muscae-domesticae*, as described by that author.

Recently Chatton and M. Léger (1911) have demonstrated the presence of an axostyle in *Leptomonas drosophilae*, and subsequently Chatton (1911) showed that the diplosome of *H. muscae-domesticae* was merely the remains of the axostyle, or axoplast. Consequently Chatton (1911) is of the opinion that the distinction between *Leptomonas* (*sensu* Chatton and Alilaire, 1908) and *Herpetomonas* is purely arbitrary, but he prefers to retain the two genera. Alexieff (1911; 1912) on the other hand assumes the identity of the two but retains the name *Herpetomonas* for the genus; moreover, he criticises Chatton and Alilaire (1908) for reviving the genus *Leptomonas* without having examined the type species.

But whichever name may be shown to be correct, it is certainly not legitimate to employ the term *Leptomonas* for all uniflagellate parasites possessing two nuclei, the kineto-nucleus being usually anterior to the tropho-nucleus. Also having shown that some species of the genus *Leptomonas*, as emended by Roubaud, may pass through a crithidial stage in their life cycle (Dunkerly, 1911) it is still less legitimate to assume that all crithidial forms occurring as true parasites of the intestines of insects should be regarded as merely representing stages in the life cycle of that genus.

On account of the fact that many trypanosomes have been shown to pass through a crithidial stage Woodcock (1909) has adopted the view that all *Crithidia* found in blood-sucking insects are stages in the life cycle of some trypanosome.

This view has been criticised by both Porter (1911) and Swingle (1911) and therefore it is unnecessary to add any further remarks on the subject. The latter author has clearly proved that *Crithidia melophagi* is a true insect parasite and not a stage in the life cycle of the sheep

trypanosome discovered by Woodcock. In addition both Porter and Patton have described the life cycles of species of *Crithidia* which do not possess any trypanosome stage and yet occur in blood-sucking insects.

Unfortunately, however, it is quite impossible at the present time to decide what form Bütschli had before him, when he described the flagellate from the gut of *Trilobus gracilis*, and which Kent made the type of his new genus *Leptomonas*. Should this parasite *L. bütschlii*, possess a crithidial stage in its life cycle, the genus *Crithidia* would have to be included as a synonym of *Leptomonas* and it would be necessary to invent a new name for the so-called *Leptomonas* forms described by Chatton and his co-workers, Mackinnon etc. At present, therefore, it is not possible to accept any final system of nomenclature, but we must wait until the life history and structure of the type species *L. bütschlii* have been worked out. Only then will it be possible to arrive at any definite conclusion on this vexed question of nomenclature.

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THE EXPERIMENTAL TRANSMISSION OF THE SPIROCHAETE OF EUROPEAN RELAPSING FEVER TO RATS AND MICE.

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(With 3 Text-figures.)

On clinical grounds alone sufficient evidence is available to postulate that European relapsing fever and Central African tick-fever are caused by different, though closely allied, organisms. However, as the spirochaetes of both diseases are morphologically identical (Schellack, 1908; Darling, 1909), I should like to record some experiments and observations I made during the winter of 1910–11 in Moscow, so as to give an exact impression of the difficulty or ease with which *Spirochaeta recurrentis* can be transmitted to mice when compared with *S. duttoni* under the same conditions. Moreover, I wish to describe some appearances in *S. recurrentis*, the exact counterpart of which have not so far been published in the case of any blood spirochaete.

For comparative purposes I also studied the behaviour in mice of strains of *S. duttoni* and *S. novyi* which were kindly given me by Prof. Martin Meyer, of Hamburg.

Previous researches on the direct transmission of *S. recurrentis* from the human being to the mouse and rat were carried out by Gabritschewsky in 1906. On this occasion he reported having transmitted the infection by passage through three animals, but no further. Mice which had withstood one infection could not be reinfected.

Following Gabritschewsky, Uhlenhuth and Haendel (1907) got *S. recurrentis* to thrive in mice and rats, after passage through a monkey. It is interesting to note that they procured the blood from Moscow, in a leech. Shortly after Uhlenhuth and Haendel's results were

published, C. Fraenkel (1907, b) also succeeded in obtaining a strain virulent for mice after passage through a monkey, the blood in this case also having been sent from Russia in a leech.

Balfour (1911) has quite lately published in tabular form an extensive and carefully compiled comparative study of the human fevers caused in different parts of the world by the various spirochaetes known at the present moment. Under the heading "Animals susceptible" we are told that the *Spirochaeta recurrentis* is transmissible to small rodents, only after passage through monkeys. This is of course the experience of Fraenkel and also of Uhlenhuth and Haendel. The experiments that are now being recorded will show that the passage through a monkey is necessary only in order to obtain a "permanent" strain.

In a synoptical review like Balfour's excellent table it is of course impossible to draw a distinction between the course of the disease in Europeans and Africans as far as regards tick-fever. On the other hand the difference is very evident in Uganda, though there it might more rightly be said to be manifested in the symptoms resulting from different degrees of acquired immunity, according to whether the affected individual comes from a tick-infested country or not. I have seen a Swahili from Mombasa and a Lendu boy from Lake Albert have most severe attacks, whilst Baganda would go about their usual occupations with numerous spirochaetes in their blood; also I have known Basesse to be bitten by ticks and not show any symptoms, though observed for a fortnight after. Of course it is possible that these last cases were bitten by non-infected ticks, especially since Hindle (1911, a) has shown that 30 % of the *Ornithodorus moubata* from Uganda are immune to infection with *S. duttoni.* However, natives from the mainland caught tick-fever in the very same rest-houses and showed numerous spirochaetes in their blood nine days later.

In crossing with a caravan of porters from a tick-free region to where the rest-houses are tick-infested, the acquisition of tick-fever soon makes itself manifest among the men, while the natives of a tick-infested region, working under the same conditions, remain unaffected.

A certain degree of immunity can therefore be acquired to this disease by repeated infections, and this immunity shows itself also in a much milder course of the disease, in the cases where the infection succeeds in overcoming the natural defence of the human organism. Accordingly we might come to the conclusion that though the

spirochaetes of Russian relapsing fever and Central African tick-fever are identical, the differences in the clinical course of the disease in the two countries might be due to the fact that the cases seen in Russia may already be partially immunised; the severity of the disease in Europeans in Central Africa on the other hand appears to be due to their being specially undefended against the spirochaete and to the special mode of transmission through the tick. Following up these suggestions, I can say that in the dozens of cases which I was able to observe in the Bassmannie Bolnitza at Moscow, the symptom complex was much less severe than that seen in any European or non-immunised African in Central Africa. The great majority of the patients in Moscow were able to stand up and make a few steps even at the height of the disease. In Africa the excruciating muscular and joint pains and the extreme weakness prevent every movement.

The personal observation of cases of tick-fever in Uganda and of relapsing fever in Moscow lead me to believe there exists conclusive clinical evidence that the disease runs a much more severe course in Africa than in Russia, and to this we can add that a distinct difference from a biological point of view between *S. recurrentis* and *S. duttoni* has been demonstrated by Uhlenhuth and Haendel, Strong (1908) and others, in so far as mice which had undergone an attack due to *S. recurrentis* spirochaetosis could be infected with *S. duttoni*, but not again with *S. recurrentis*.

From what I have seen in Moscow I am of the opinion that the transmission of relapsing fever in Russia, is commonly effected by *Pediculus vestimentorum* and not by *Cimex*.

Mackie (1907) appears to have been the first to incriminate pediculi, later on Manteufel (1908, b), Smith (1910), Sergent and Foley (1910), and Fehrmann (1910) have brought forward experimental and clinical evidence in support of this view.

My reasons are so far purely clinical. Having visited the big municipal Night Hostels at Moscow I soon noticed that they were kept with such scrupulous cleanliness, disinfected so lavishly, the beds being of iron, the floor cemented, that it was not possible for bed-bugs to thrive to any extent on the premises. The people sleeping there were allowed, however, to go to sleep in their own clothes. The introduction of these model homes had not had any effect on the incidence of relapsing fever, for the places were still hotbeds of the disease during winter. On the other hand, though I changed my rooms several times I found bugs in every successive lodging, and as I was told in Moscow

this can hardly be avoided. Yet no foreigner, or Russian of a better class, ever catches relapsing fever. To this may be added the fact that when I asked for clothes-lice and promised to pay a kopek for two, the attendants from the Night Hostel brought me next morning a small ounce bottle crammed with *Pediculus capitis* and *P. vestimentorum* collected off the sleepers. If relapsing fever were transmitted by bed-bugs it would be much more disseminated than it is at present in Moscow.

In Balfour's synoptical table a morphological distinction appears to be made between the spirochaetes of European, Central African and American relapsing fever and the spirochaetes of the Egyptian, Algerian and Asiatic relapsing fevers, from the observation of Fraenkel that the former have peritrichous flagella, whilst nothing is known about their presence in the latter.

As far as my personal observations go, I have often seen a terminal filament in *S. recurrentis* when using dark-ground illumination, whilst so far the "flagella" have only been seen in dry films, which have been specially prepared. I believe that the peritrichous "flagella" shown in Fraenkel's (1907) and also Zettnow's (1906) excellent photographs are threads of periplast, as Schellack (1908) and Karwacki (1912) suggest.

Another explanation of the undeniable appearances in the specimens of the authors who consider flagella to be an integral part of spirochaetes, is that they may be rudimentary structures similar to the "ribs" seen in the "undulating membrane" of *Spirochaeta* (*Cristispira*) *balbianii*; it is known that Fantham (1909) found in the crystalline style of *Tapes aureus* a continuous series of long and short, narrow and broad forms; also spirochaetes with sharply pointed, tapering or rounded ends and others whose two ends were unequally pointed or rounded. A glance at Fantham's figures shows all gradations in shape from the typical *Cristispira balbianii* to the spirochaete as seen in the blood of mammals. Therefore it is possible that these "flagellar structures" in *S. recurrentis*, *duttoni* and *novyi* may be similar to those seen in a much more developed form in the spirochaetes of oysters and mussels.

Regarding the terminal filament it appears to be a common feature of many spirochaetes. It has been seen repeatedly in *Treponema pallidum*, also in Noguchi's (1911) cultures of this parasite, by Schellack (1908) in *S. recurrentis*, by Prowazek and Hoffman in *S. balanitidis*, in *S. dentium* by Hartmann and Mühlens, by Zettnow

(1906) in *S. duttoni*, and by Novy and Knapp (1906) in *S. duttoni* and *novyi*.

This terminal filament is not visible in all specimens of *S. recurrentis, duttoni*, or *novyi*. It is quite possible that Schellack is right in considering it only present in young specimens, which have but lately divided. This would be an observation more in favour of the transverse division of spirochaetes, which moreover can often be seen taking place in blood-films if patiently examined by dark-ground illumination. Miss Mackinnon (1909, a) observed in two cases apparent longitudinal division but in eleven the division was apparently transverse. It could not be said with certainty, except in one instance, that these transverse divisions were not the final act in a longitudinal division. Therefore it is apparent that if both modes of division occur, the transverse is by far the more common in blood. The same seems to hold good for the divisions of *Treponema pallidum* observed in Noguchi's cultures.

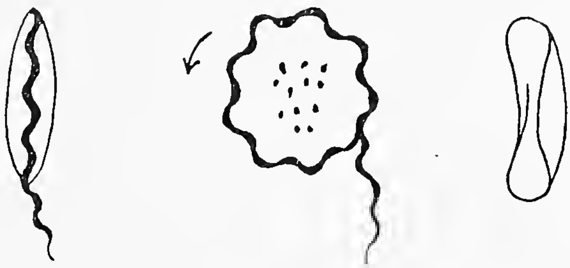

Fig. 1. Fig. 2. Fig. 3.

Fig. 1. Side view of ring.
Fig. 2. Front view. The arrow indicates the direction of rotation.
Fig. 3. Erythrocyte of mouse, to show comparative size.

On comparing my notes on the three strains of spirochaetes which I studied, I cannot say that I noticed any special mode of progression which was typical for any one species. The screw-like and the lashing, trypanosome-like movements, as also more or less flattened spirals, occurred in all the strains according to circumstances and the degree of infection in the animals experimented upon.

A most peculiar appearance in *S. recurrentis* was seen on several occasions in the blood of mice which had been infected with relapsing fever blood. It consisted in the rolling up or encircling of the spirochaete round what appeared to be an exceedingly thin membrane containing small refringent granules, which were actively moving. It was difficult to recognise these forms as spirochaetes, because an

exceedingly rapid circular movement was maintained, which gave the whole the appearance of a faintly visible ring. It was only when the rotation stopped for a second or so that the spirillar structure could be seen and the short appendix I have drawn be detected. On a few occasions I succeeded in seeing the organism sideways and then it appeared to me that it was not only rotating in a circle, but also around its own axis. The membrane I have already mentioned could then be made out as a very faint outline. It is an integral part of the spirochaete and I do not think that it can be considered to be a laked erythrocyte.

Similar but smaller bodies have been described by Butler in *S. duttoni* when kept outside the body. Globose swellings have been seen by Carter in the spirochaetes occurring in Southern Arabia. Schellack (1908) also describes similar appearances in *S. novyi* after washing them in saline solution; Karwacki observed the rolling up into a spiral of *S. recurrentis* in relapsing fever blood kept in a leech.

Fantham's (1911, p. 491) observations on these features, or at least somewhat similar ones, are of interest.

> "Encystment of Spirochaetes has been described by several writers. Up to the present, neither in the Spirochaetes of the blood nor in those of Lamellibranchs have I found true encystment. Two types of pseudo-cyst forms have, however, been encountered:
>
> 1. The Spirochaete becomes more closely coiled, either about its centre or nearer one end, so that a ball-like form is produced. This ball simulates a cyst with the body of the Spirochaete protruding from either end....But this form is only temporary, the Spirochaete uncoiling after a short time and swimming away normally.
>
> 2. Plasmatic cysts may be formed. Here the Spirochaete is dying, and so is not normal. The periplast relaxes and the cytoplasm tends to collect into small irregular masses or droplets, which cause local bulgings along the body. I have not seen these protoplasmatic aggregations other than in animals in an almost moribund condition, when the Spirochaetes, naturally, were under unfavourable conditions.... One or two similar plasmatic cysts may occasionally be found under similar conditions on a blood Spirochaete."

It will be seen from these descriptions that the condition of which I give a figure is somewhat different from anything already observed and occurs moreover in a blood spirochaete, in an animal not moribund, but more probably as a result of the action of the anti-bodies of the host. As Fantham suggests, these rings may be a special degeneration-form, due however in this special case to the immune bodies in the mouse causing the spirochaetes of relapsing fever to die out. At the time

I first saw them, I considered them more in the light of a resting stage.

I saw and demonstrated to various workers in the Laboratory at Moscow the spirochaetes straightening themselves out, after having been for several minutes unrecognisable in their circular form. After straightening out, they move as usual for a few seconds and then return to their circular shape again. I was unable to make out what became of the membrane in the meanwhile, the granules were however scattered.

The whole structure cannot be stained and can only be examined in the living state. I am also unable to say whether this condition bears any special relation to the granular stage of spirochaetes, as described in the papers of Leishman (1910), Balfour (1908), and Hindle (1911, c).

I have only seen it in *S. recurrentis*. In *S. duttoni* and *novyi* I only saw the usual well-known small nodules which have so often been described and which are a common appearance after treatment with weak saline or tap water and which, of course, are much smaller.

Turning now to the results of my experiments on animals I may mention that all blood examinations were made with Leitz's dark-ground condenser. In most cases the blood was taken from the arm-vein of the patient with a syringe and immediately defibrinated. On two occasions blood was taken in leeches. After two days this blood was still infective for mice, but not later.

I should like to express my thanks to Drs Remenzow and Smirnow of the Basmannie Bolnitza for the help they gave me.

Protocols.

I. *Unsuccessful attempts to infect animals with spirochaetosis (Russian strain).*

Exps. 1–3. 16th Nov. 1910. Three mice injected intra-peritoneally with 0·5 c.c. human blood containing numerous spirochaetes (relapsing fever) directly from the patient.

The blood of the three mice was examined with negative results on the 17th, 18th, 19th, 20th, 22nd, 24th, 27th, 29th Nov. and 2nd Dec. 1910. The animals were then killed.

Exp. 4. 18th Nov. 1910. Rabbit injected intravenously with 3 c.c. of defibrinated relapsing fever blood.

Examined with negative results on the 19th, 20th, 25th and 27th Nov. 1910. No further examination.

Exps. 5, 6. 18th Nov. 1910. Guinea-pig injected intra-peritoneally with 2 c.c. of defibrinated relapsing fever blood.

Examined with negative results on the 19th, 20th, 22nd, 24th, 25th and 26th Nov. 1910.

1st Dec. 1910. Intra-peritoneal injection of 1 c.c. of defibrinated relapsing fever blood. Examined on next two days with negative results.

Exp. 7. 18th Nov. 1910. White rat, intra-peritoneal injection of 1·5 c.c. of defibrinated relapsing fever blood, diluted with an equal quantity of sterile saline, two hours after having been taken from patient.

Examined with negative results on the 19th, 20th, 22nd, 24th and 26th Nov. 1910.

Exps. 8–10. 18th Nov. 1910. White rat, injected intra-peritoneally with 1·5 c.c. defibrinated relapsing fever blood.

Examined with negative results on the 19th, 20th, 22nd, 24th and 25th Nov. On this date it was re-injected, also intra-peritoneally, with 1 c.c. of defibrinated relapsing fever blood diluted with 2 c.c. of sterile saline.

Examined with negative results on the 26th, 27th and 29th Nov.

1st Dec. 1910. Injected intra-peritoneally with 1 c.c. of defibrinated relapsing fever blood diluted with 2 c.c. of sterile saline.

Examined with negative results on the next two days.

Exp. 11. 18th Nov. 1910. White rat injected intra-peritoneally with 1·5 c.c. of defibrinated relapsing fever blood.

Examined with negative results on the 19th, 20th, 22nd and 24th Nov. 1910.

Exp. 12. 19th Nov. 1910. Mouse, injected intra-peritoneally with 0·5 c.c. of defibrinated relapsing fever blood, which had been kept 24 hours on ice.

Examined with negative results on the 20th and 22nd Nov. Died next day.

Exps. 13–15. 25th Nov. 1910. Three mice; each received an intra-peritoneal injection of 0·5 c.c. of defibrinated relapsing fever blood in 1·5 c.c. saline.

Examined with negative results on the 26th, 27th, 29th Nov. and 2nd Dec. 1910.

Exps. 16–19. 1st Dec. 1910. Four mice each injected intra-peritoneally with 0·5 c.c. defibrinated relapsing fever blood in 0·5 c.c. saline.

Examined with negative results the next and following day.

It might be argued from the foregoing experiments that the negative results of the inoculations were due to the animals being immune. This was not the case, because the animals of experiments Nos. 7, 8–10, 11, 13, 14, 15 proved themselves susceptible to injections carried out later on, as the following experiments will show.

II. *Experiments in which infection succeeded only after repeated attempts.*

Exp. 20. Same rat as in Exp. No. 7. 2nd Dec. 1910. Intra-peritoneal injection of 0·3 c.c. defibrinated relapsing fever blood in 0·7 c.c. saline.

The next day a few spirochaetes were seen in the blood, but they had disappeared the following day.

EXP. 21. Same rat as in Exps. Nos. 8–10 in the previous table; had therefore been unsuccessfully inoculated three times with relapsing fever blood. 14th Dec. 1910. Intra-peritoneal injection of 0·3 c.c. relapsing fever serum containing numerous spirochaetes.

15th Dec. 1910. Numerous "circular" forms seen.

16th Dec. 1910. Spirochaetes still numerous.

Next day negative.

EXP. 22. Same rat as in Exp. No. 11; it had therefore been fruitlessly injected once with relapsing fever blood and once with blood from a mouse containing numerous spirochaetes.

14th Dec. 1910. Intra-peritoneal injection of 0·3 c.c. of defibrinated relapsing fever blood.

A few spirochaetes were seen next day, but had disappeared 24 hours after.

EXP. 23. Same mouse as in Exp. No. 13. 14th Dec. 1910. Intra-peritoneal injection of 0·3 c.c. defibrinated relapsing fever blood in 1·5 c.c. saline.

Spirochaetes detected next and following day.

EXP. 24. Same mouse as in Exp. No. 14. 14th Dec. 1910. Intra-peritoneal injection of 0·3 c.c. defibrinated relapsing fever blood in 0·5 c.c. saline.

15th Dec. 1910. Numerous winding circular forms present.

16th Nov. 1910. Negative.

EXP. 25. Same mouse as in Exp. No. 15. 14th Dec. 1910. Intra-peritoneal injection of 0·3 c.c. of defibrinated relapsing fever blood in 0·5 c.c. saline.

15th Dec. 1910. Spirochaetes with peculiar, lashing, trypanosome-like movements.

16th Dec. 1910. Negative.

Marked natural immunity was therefore not present in these animals, and the previous unsuccessful attempts may well have been due to the strains injected being less virulent than the later ones.

The blood injected on the 14th Dec. 1910 was the most infective of all. It came from a patient who died the next day. He had spirochaetes in his blood during nine days and suffered from marked icterus; his temperature, however, did not rise above 39° C. The blood contained about 10 to 15 spirochaetes in every $\frac{1}{12}''$ oil immersion field.

III. *Experiments in which infection followed the first inoculation.*

EXP. 26. 18th Nov. 1910. White rat injected intra-peritoneally with 1·5 c.c. defibrinated relapsing fever blood.

19th Nov. 1910. Spirochaetes numerous.

Examined with negative results on the 20th, 22nd, 24th, 26th, 27th and 29th Nov. 1910.

EXPS. 27, 28. 19th Nov. 1910. Two mice; intra-peritoneal injection of 0·5 c.c. defibrinated relapsing fever blood, which had been kept 24 hours in the ice-chest.

20th Nov. 1910. Spirochaetes found in both animals.

Examined with negative results on the 22nd, 24th, 26th, 27th, 29th Nov. and 2nd Dec. 1910.

Exp. 29. 25th Nov. 1910. Mouse; intra-peritoneal injection of 0·5 c.c. of defibrinated relapsing fever blood in 1·5 c.c. saline.

26th Nov. 1910. Spirochaetes detected; mouse killed and blood injected into mice of Exps. Nos. 50, 51.

Exps. 30, 31. 25th Nov. 1910. Two mice; intra-peritoneal injection of 0·5 c.c. defibrinated relapsing fever blood in 1·5 c.c. saline.

Spirochaetes seen on two following days. Negative the 29th Nov. and 2nd Dec. 1910.

Exps. 32–34. 1st Dec. 1910. Three white rats; intra-peritoneal injection of 0·5 c.c. relapsing fever blood in 1·5 c.c. saline.

All three showed spirochaetes in their blood the next day and were negative the following day.

Exp. 35. 1st Dec. 1910. Mouse; intra-peritoneal injection of 0·5 c.c. of defibrinated relapsing fever blood in 0·5 c.c. saline.

Spirochaetes found next, but none the following day.

Exps. 36–40. 8th Dec. 1910. Five mice; intra-peritoneal injection of 0·3 c.c. defibrinated relapsing fever blood in 0·5 c.c. saline.

One showed spirochaetes next day only.

Spirochaetes found in all four animals the next two days, but none on the third day.

IV. *Experiments in which infection followed the first and subsequently repeated inoculations.*

Exp. 41. Same mouse as in Exp. No. 27. 14th Dec. 1910. Intra-peritoneal injection of 0·15 c.c. defibrinated relapsing fever blood in 0·5 c.c. saline.

15th Dec. 1910. Spirochaetes detected.

16th Dec. 1910. Atypical, short spirilla seen, possibly bacteria (?).

17th Dec. 1910. Negative.

Exp. 42. Same mouse as in Exp. No. 28. 14th Dec. 1910. Intra-peritoneal injection of 0·15 c.c. defibrinated relapsing fever blood in 0·5 c.c. saline.

15th Dec. 1910. Spirochaetes present.

16th Dec. 1910. Negative.

Exp. 43. Same mouse as in Exp. No. 31. 14th Dec. 1910. Intra-peritoneal injection of 0·3 c.c. defibrinated relapsing fever blood in 0·5 c.c. saline.

15th Dec. 1910. Spirochaetes detected with peculiar lashing, trypanosome-like movements. Negative next day.

Exp. 44. Same mouse as in Exp. No. 36. 14th Dec. 1910. Intra-peritoneal injection of 0·3 c.c. of defibrinated relapsing fever blood in 0·5 c.c. saline.

Spirochaetes found the two following days. Mouse was killed and blood used for Exps. Nos. 56, 57.

EXPS. 45, 46. Same mice as in Exps. 37, 38. 14th Dec. 1910. Intra-peritoneal injection of 0·3 c.c. defibrinated relapsing fever blood in 0·5 c.c. saline.

Next day numerous "lashing" forms were seen in the blood of both.

The day after the spirochaetes had disappeared.

EXPS. 47, 48. 14th Dec. 1910. Subcutaneous injection of 0·3 c.c. defibrinated relapsing fever blood in 0·5 c.c. saline.

The next two days numerous spirochaetes detected. None seen next day.

The foregoing experiments show that mice can be reinfected by a second inoculation made up to 25 days after the first successful inoculation.

Attempts to induce a permanent infection.

EXP. 49. 26th Nov. 1910. Guinea-pig, injected intra-peritoneally with 0·1 c.c. mouse-blood from Exp. No. 29. Examined on two following days with negative results.

EXPS. 50, 51, 50*a*, 51*a*. 26th Nov. 1910. Two mice, intra-peritoneal injection of 0·3 c.c. blood containing numerous spirochaetes from mouse No. 29, diluted in 0·5 c.c. saline.

Examined with negative results on the 27th, 29th Nov. and 2nd Dec. 1910.

To exclude the possibility of these two mice being immune, they were injected on the 8th Dec. 1910 with 0·3 c.c. defibrinated relapsing fever blood in 0·5 c.c. saline.

Spirochaetes were found in the blood of both on two following days.

EXPS. 52, 52*a*. 29th Nov. 1910. White rat, intra-peritoneal injection of 0·5 c.c. of blood of mouse showing spirochaetes.

Examined the following three days with negative results.

Also in this case the rat was injected on the 2nd Dec. 1910, with 0·3 c.c. defibrinated relapsing fever blood and showed next day the spirochaetes in its blood, but not the day after.

EXPS. 53, 53*a*. Same rat as in Exp. 11. 29th Nov. 1910. Intra-peritoneal injection of 1 c.c. of defibrinated mouse blood showing spirochaetes.

Examined with negative results on the 30th Nov. and 2nd Dec. 1910. The injection of 0·3 c.c. relapsing fever blood on the 14th Dec. gave a positive result next day. No spirochaetes found the day after that.

EXPS. 54, 55, 54*a*, 55*a*. 26th Nov. 1910. Two mice, intra-peritoneal injection of 0·5 c.c. blood from Exp. No. 29.

Examined with negative results on the 27th, 29th Nov. and 2nd Dec. 1910.

8th Dec. 1910. Both were injected with 0·3 c.c. defibrinated relapsing fever blood and showed spirochaetes in the blood the next and following days.

EXPS. 56, 57. 16th Dec. 1910. Two mice injected intra-peritoneally with blood from mouse in Exp. No. 44.

One showed a few spirochaetes next day. It was killed and its blood injected into another mouse, with negative results. The other mouse did not show any spirochaetes at all, it was examined on three days.

These experiments tend to show that in order to obtain a permanent strain of Russian spirochaetosis, capable of being easily transmitted from one mouse or rat to another, it is first neeessary to pass the organism through a monkey, as has been done by Uhlenhuth, Haendel, and Fraenkel.

CONCLUSIONS.

(1) European relapsing fever, caused by *Spirochaeta recurrentis*, can be transmitted directly to mice and rats. The infection does not appear to last more than two days (Gabritschewsky).

(2) As open and spirillar forms, corkscrew shapes and lashing spirochaetes with trypanosome-like movements, according to different circumstances, may appear in all the three strains I have observed, there is no possibility so far of distinguishing morphologically *S. recurrentis* from *duttoni*, or *novyi*.

(3) The marked distinguishing feature between Russian relapsing fever and Central African tiek-fever is eaused by the vague factor called "virulence," and this is decidedly greater in the case of *S. duttoni*.

(4) On several oecasions it was possible to reinfect rats and miee with quite small injections of relapsing fever blood. A marked immunity as a result of previous infections was therefore not present in these cases.

(5) In Moscow, relapsing fever appears to be transmitted by *Pediculus vestimentorum*.

(6) Peculiar ring forms, containing granules, were deteeted in miee on several occasions, possibly as a result of beginning degeneration in *S. recurrentis*.

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NOTE ON *GNATHOSTOMUM SPINIGERUM.*

By S. N. MITTER, G.Beng.V.C.,
Lecturer on Pathology, Royal Veterinary College.

(From the Raymond Research Laboratory, Calcutta.)

(With Plate V.)

Since I published my note on the occurrence of *Gnathostomum spinigerum* in the stomach of a domestic cat (*Journ. Tropical Vet. Sci.*) two other cases have been observed in this Laboratory.

(A) While conducting an autopsy on a fox-terrier dog, born and bred in this country, two specimens of *Gnathostomum* (♂ and ♀) were found free in the peritoneal cavity. On opening the stomach, a hard tumour with a sinus in the centre was noticed from which a third *Gnathostomum* protruded into the cavity of the stomach. The sinus extended through the tumour (?) to the peritoneal cavity. From the peritonal end of the sinus a fourth *Gnathostomum* was protruding.

(B) The carcass of a leopard (*Felis pardus*) was received from the Calcutta Zoological Garden for post mortem examination and report. The Superintendent informed us that the animal had been ailing for two or three days. Two other leopards in the same garden had died within two or three days with similiar symptoms. These animals were not however sent for autopsy.

On opening the stomach two suppurating tumours (Plate V) of the size of large walnuts discharging semi-liquid pus were found in the mucous membrane. In the contents of the stomach, chiefly mucous, were found several free *Gnathostoma*, and eighteen specimens were found in the tumours. The peritoneal cavity also contained several specimens which had no doubt escaped from the stomach through the sinuses leading to the peritoneum. Death was due to perforation of the stomach wall and gastritis caused by these parasites.

This species has already been found in the wild cat (*F. catus*), in the puma (*F. concolor*) and in the tiger (*F. tigris*).

Portion of the mucous membrane of the stomach of a leopard showing two tumours caused by *Gnathostomum spinigerum*.

Specimen prepared by S. N. Mitter, Bengal Veterinary College.

Adelchi Negri

Pavia 12 Febbraio 1910

IN MEMORIAM

ADELCHI NEGRI.

Born 16 July, 1876 at Perugia.
Died 19 February, 1912 at Pavia.

(With Portrait, Plate VI.)

It is with great regret that we record the early death of Adelchi Negri, for a career full of promise has been cut short and his loss is one that will be felt far beyond the confines of Italy.

Negri was born in Perugia on the 16th of July, 1876, being the only child of Professor Cav. Raffaele Negri by his wife Emilia née Almici. He pursued his medical studies in Pavia where he became qualified in 1900 being afterwards assistant to Golgi at the Pathological Institute. In 1905 he became libero docente in general pathology. On the 16th of September, 1905, he married Signorina Lina Luzzani who shortly before had graduated in medicine in Pavia. In 1908 he was made Professor of Microbiology in the University of Pavia, a position he occupied until his death which took place on the 19th of February, 1912. He left no children. His talented widow and his aged parents, the latter now residing in the Province of Brescia, survive to mourn his loss.

Negri's earlier publications (1899–1902) relate to the structure of the red blood-corpuscles, the origin of blood platelets, the cytology of gland cells in mammalia and the changes undergone by blood elements during coagulation. In these papers he already showed his ability as an original investigator. In 1910 he published a paper upon cell regeneration in parathyroid glands.

It was, however, in 1903 that Negri published his first contribution to the study of rabies, a disease with which his name will always remain associated. To all who are familiar with this disease the "Negri bodies" will be equally familiar, for, dating from his initial

discovery of these bodies the latter have come to be regarded as of prime importance in the diagnosis of rabies. Negri demonstrated successively the constant presence of these bodies in the nervous system of man, rabbits, dogs, and various other animals affected with rabies. From the start he regarded the bodies as parasitic Protozoa, and he subsequently traced out with much patient labour the supposed cycle of development of *Neuroryctes hydrophobiae* Calkins. He devoted nearly ten years of his life to the study of rabies.

Although his chief contributions related to rabies, he also turned his attention to other subjects. He worked upon bacillary dysentery, and was the first to show that vaccine virus traverses bacterial (Berkefeld) filters.

He, moreover, made important observations upon *Haemoproteus*, and especially upon *Sarcocystis muris* (Blanchard) Labbé. Although he was forestalled by Theobald Smith (1905), who successfully transmitted *S. muris* by feeding healthy mice upon the muscles of infected mice, he went further in that he transmitted the parasite in a similar manner from affected rats to healthy rats and guinea-pigs. He proved thereby that one species of *Sarcocystis* may occur in different species of hosts, an observation which throws grave doubt upon the validity of numerous so-called species of *Sarcocystis* which have been described from different species of animals. Doubts as to the validity of some of these species are, moreover, justified by Negri's finding that *S. muris* differs in its morphology according as it occurs in the rat or guinea-pig. It should be mentioned, in this connection, that Negri's views regarding the cycle of development of this parasite differ from those advanced by other authors, and they appear justified in the light of his painstaking experimental work.

During the last three years of his life Negri took a very active part in the campaign against malaria in Lombardy concerning which he issued two reports.

Negri carried on his work for years with unabated zeal and enthusiasm in spite of increasing infirmity, and he won the regard and admiration of all who knew him. Although the writer had not the privilege of knowing him personally, a correspondence extending over several years confirmed him in the belief that Negri was one of the most obliging of men, and it is with a keen sense of the loss which science has sustained through his premature death that this tribute is offered to his memory.

G. H. F. N.

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—— (1906). Ueber Filtration des Vaccinevirus. *Zeitschr. f. Hyg.* LIV. 327–346 (same as the preceding).

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—— (1908, 1910). Osservazioni sui Sarcosporidi. Nota I. *Rendiconti R. Accad. Lincei* XVII. 1° Semestre, Ser. V., pp. 561–567; Nota II, *Ibid.* pp. 666–677; Nota III, *Archives per le sci. med.* 1910, XXXIV.

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BOOKS.

ASHFORD, B. K. and IGARAVIDEZ, P. G. (1911). *Uncinariasis (Hookworm Disease) in Porto Rico:* A Medical and Economic Problem. Washington: Government Printing Office, U.S. America. Document No. 808. 335 pp. with a map and many illustrations.

An exhaustive report dealing with conditions affecting Uncinariasis in Porto Rico, the history of a 10-years' campaign against the disease and clinical studies thereon. Pages 99–238 contain a compilation of the Reports of the Porto Rico Anaemia Commission for the years 1904–09 inclusive. A summary of the forthcoming report for 1909–10 then follows, together with a description of methods of combating Uncinariasis. An Appendix contains the case histories of 306 patients who were made the subject of special study. The report is very interesting but the English is shocking. N.

BAHR, P. H. (1912). *Filariasis and Elephantiasis in Fiji.* Being a report to the London School of Tropical Medicine. London: Witherby & Co. 326 High Holborn, W.C. *Journ. Lond. Sch. Trop. Med.* Suppl. No. 1. 192 pp. with many coloured and monochrome plates, numerous charts and one map.

In this report the author gives an account of his investigations on Filariasis and Elephantiasis in Fiji during the year 1910. The extreme frequency of Filariasis in this region enabled many interesting results to be obtained, and Dr Bahr is to be congratulated upon the manner in which they are presented to the reader. The *Filaria* occurring in Fiji, although exhibiting no periodicity in its presence in the blood, is shown to be indistinguishable on morphological grounds from the common *Filaria bancrofti.* It is uncertain, therefore, whether the *Filaria* of Fiji should be regarded as a new species or not. *Stegomyia pseudoscutellaris* (Theob.) is shown to be the chief agent in transmitting the disease, *Culex fatigans* being a much less efficient intermediary. The author gives some interesting photographs showing the development of the *Filaria* inside the mosquito, and also the worms escaping from the proboscis.

It is suggested that the comparative sterility of the Fijians is due to the extreme prevalence of filariasis amongst the natives, as this disease often attacks the male genitalia and the worms are frequently found in the testes. Elephantiasis of the scrotum is very common, and some of the cases are illustrated by photographs. The appendices occurring at the end of the report contain details of experiments and also many interesting notes. Of especial interest is the description of the breeding habits of *Stegomyia pseudoscutellaris.*

This insect develops most readily in 0·75 % normal saline; and when jars of fresh water, normal saline, and sea-water, respectively, are placed in the mosquito cages, the insects lay most of their eggs on the jar containing the normal saline. This species is thus well adapted for living in islands where only brackish water is present. H.

Howard, L. O. (1911). *The House-fly disease carrier*, an account of its dangerous activities and of the means of destroying it. New York: F. A. Stokes Company. 312 pp., 41 illustrations. 21 × 14 cm. Price $1.60 net, cloth.

Whilst avowedly popular in character, this book may be consulted with great profit by the scientific reader, since it contains much useful information gathered from scattered publications and presented in a condensed and convenient form. The illustrations, many of which are original, are in some cases of extraordinary merit. The zoological position, life-history and habits of *Musca domestica* Linn. form the subjects of the first chapter, and the chapters that follow deal with the natural enemies of the house-fly; the part flies play as disease-carriers; remedies and preventive measures directed against house-flies; other species of flies which frequent houses. A good bibliography and five appendices conclude the work. The appendices relate severally to: flies frequenting human dejecta and found in kitchens; flies reared from cow manure; Health Department regulations against flies in the United States; directions for building a sanitary privy; a simple apparatus for use in the safe disposal of night-soil.

The author and publishers are to be congratulated upon this excellent book, which is the best presentation of the subject which has hitherto appeared. N.

Klieneberger, C. and Carl, W. (1912). *Die Blut-Morphologie der Laboratoriums-Tiere.* 109 pp., 14 coloured plates with legends. 24 × 16 cm. Leipzig: Johann Ambrosius Barth. Price unbound, 10 marks.

This beautifully illustrated little work should prove most useful to those working upon the comparative morphology of the blood and upon Haematozoa. The authors have not confined their attention merely to the blood of ordinary laboratory animals (mouse, rat, guinea-pig, rabbit, cat and dog) for they also fully illustrate and describe the blood of other animals which are not so commonly examined (hedgehog, sheep, monkey, fowl, pigeon and frog). The studies carried out by the authors upon the various kinds of blood were carefully conducted, having regard to the health of the animals from which the samples were taken. Due care moreover was taken to examine the blood in animals under varying conditions, *i.e.* during hibernation, gestation or when in heat. The blood was examined according to the usual clinical methods. Haemoglobin determinations were made with Sahli's apparatus, blood counts with the Thoma-Zeiss apparatus. The blood elements were examined in films and sections stained in a variety of ways.

Taking the blood of the white mouse as an example, the authors state how samples thereof should be taken, and describe the variations in the proportions of the various elements in different parts of the body and under different conditions in animals fed and unfed. They conclude the description with a tabular statement containing average figures for Haemoglobin content, and the proportionate numbers of the various blood elements.

The authors and publishers are to be congratulated upon the excellence of the work. It is to be hoped that in a future edition the authors may see their way to add descriptions and figures of other species of blood to what they have already given in the book under review. We heartily recommend the book to the notice of our readers. N.

MATHIS, C. and LEGER, M. (1911). *Recherches de Parasitologie et de Pathologie humaines et animales au Tonkin.* viii + 451 pp., 14 coloured plates and many text-figures. 25 × 17 cm. Paris: Masson et Cie.

This book constitutes a valuable contribution to our knowledge of human and animal diseases occurring in and about Tonking, for it describes the results of investigations carried out by the authors since 1908. During this period the authors have published a number of papers in various French journals and these papers have been incorporated in the book, together with a remarkably fine series of hitherto unpublished illustrations. *Part I* (pp. 1–256) deals with human diseases: Malaria and the Anophelines of Tonking; relapsing fever; beri-beri; Helminthiasis, giving the results of the examination of the stools in 1250 healthy natives and of 200 healthy Europeans; intestinal flagellates; amoebic dysentery and hepatitis; Filariasis in northern Indo-China. *Part II* (pp. 257–430) deals with animal diseases: the malarial parasites of monkeys; the description of species of *Haemoproteus*, *Leucocytozoon*, *Haemogregarina*, *Piroplasma*, *Trypanosoma*, *Trypanoplasma; Spirochaeta* in the rabbit; *Filaria*, etc. A classified list of animals examined, together with their parasites, concludes the section.

Malaria (tertian, quartan and tropical) is irregularly distributed in Tonking, the endemic index averaging 6·63. Fifteen species of Anophelines (none new) are described, and their relation to malaria considered. The authors infected monkeys and mice with the spirochaetes obtained from human cases of relapsing fever, but failed to transmit infection by means of lice. A new flagellate, *Prowazekia weinbergi*, occurring in diarrhoeic stools, is described, the parasite having been successfully cultivated with bacteria and its development followed. The most interesting sections of Part II are those dealing with *Leucocytozoa* and *Trypanosoma*. It is sad that the authors' beautiful figures are reproduced by the three-colour process upon the perishable glazed paper which is so commonly used nowadays, a paper which should be discarded in publishing a work of such high merit. N.

NEVEU-LEMAIRE, M. (1912). *Parasitologie des Animaux Domestiques*, maladies parasitaires non-bactériennes. 1257 pp., 770 text-figures. 18 × 14 cm. Paris: J. Lamarre et Cie, 4 Rue Antoine Dubois (VI^e^). Price 16 francs, cloth.

This work is intended for the use of parasitologists, zoologists and veterinarians in particular. It covers a very wide field, dealing with parasitology both in temperate and tropical countries. Part I (pp. 15–182) deals with vegetable parasites, Part II (pp. 183 *et seq.*) with animal parasites: Protozoa, Vermes, and Arthropoda. At the end of the volume there occur (*a*) a very complete list of the parasites of domesticated animals, the parasites being arranged according to the situations in which they occur upon the host, (*b*) a short general bibliography, and (*c*) full indices to the subject-matter. Many references to the literature are given in footnotes throughout the book. The

illustrations are very numerous and fairly good. On the whole the book should fill a present want. N.

PROWAZEK, S. VON (1911). *Handbuch der Pathogenen Protozoen.* (1–3 Lieferung.) Leipzig: Verlag von J. A. Barth. Pp. 1–117, 119–248, 249–360. 6 plates (4 coloured) and numerous text-figures. Prices of parts (unbound), respectively 6.40 M., 7.20 M. and 6 M.

The work before us represents the combined work of numerous authors. *Part I* contains: An Introduction by R. Nocht; Fixing and Staining methods by E. Giemsa; Classification of Protozoa by M. Hartmann; The Dysentery Amoebae by M. Hartmann; *Entamoeba coli* by H. Werner; Flagellates (*Trichomonas*, *Lamblia*) by E. Rodenwaldt; *Costia necatrix* by E. Neresheimer; The genus *Trypanoplasma* by E. Neresheimer. *Part II* contains: "The Chlamydozoa" by S. v. Prowazek and B. Lipschütz, this section including: Vaccinia, Variola, Virus myxomatosum and "jaundice" in silkworms, by S. v. Prowazek; Epitheliosis desquamativa conjunctivae by A. Leber and v. Prowazek; Trachoma and chlamydozoal diseases of the mucous membranes by L. Halberstaedter; Rabies by Maresch; Molluscum contagiosum by B. Lipschütz; Epithelioma contagiosum of Fowls by B. Lipschütz. *Part III* contains: Pathogenic Trypanosomes by M. Mayer; Cnidosporidia (Myxo- and Microsporidia) by O. Schröder; Sarcosporidia by E. Teichmann. When completed the book will contain upwards of 500 pages.

It will be seen from the foregoing statement regarding the contents of the work that it departs from the usual order adopted by authors in presenting a subject to readers. There is really no special order about the book—it is composed of a series of disconnected treatises in which an attempt is made, with more or less success, to summarize our present knowledge. Some of the views of Hartmann on classification are by no means likely to meet with general acceptance. The "Chlamydozoa," about which there has been much controversy, are treated very fully. Some of the authors base their opinions to a great extent upon ingenious hypotheses and "Hilfshypothesen," consequently the book is scarcely one to be commended to workers commencing the study of protozoology. The work is, nevertheless, valuable for the many suggestions it contains—suggestions which will doubtless influence future investigations. The text is excellently illustrated, and copious references are given to the literature. N.

ROSS, E. H. (1911). *The reduction of domestic mosquitos.* Instructions for the use of municipalities, town councils, health officers, sanitary inspectors and residents in warm climates. London: John Murray, Albemarle Street, W. 114 pp. with 18 plates. 23 × 15 cm. Price 5/- net.

As stated in the introduction, the object of this book is to bring to public notice the necessity for the reduction of domestic mosquitoes and to describe how this is best effected. The book is consequently not overburdened with detail and written with a view to familiarizing those in authority with the practical importance of mosquito reduction. The twelve chapters into which the book is divided deal with the importance of domestic mosquitoes and their biology; the fever census; estimating the cost of mosquito reduction; the manner of starting anti-mosquito campaigns and observing their effect.

MISCELLANEOUS.

DEEKS, W. E., and JAMES, W. M. (1911). *A Report on Hemoglobinuric Fever in the Canal Zone: a study of its etiology and treatment.* 177 pp. Canal Zone: Isthmian Canal Commission Press, Quartermaster's Department, Mount Hope.

Fourth Report (1911) *of the Wellcome Tropical Research Laboratories at the Gordon Memorial College, Khartoum* (A. Balfour, Director). Vol. *A*—Medical. 404 pp. with 14 coloured plates and 160 text-figs., maps and plans. Vol. *B*—General Science. 333 pp. with 9 coloured plates and 180 text-figs., maps and plans. London: Baillière, Tindall & Cox, 8, Henrietta Street, Covent Garden. 28 × 20 cm. Price 18/-.

Contents of *Volume A*—Medical. Introduction—Changes and Plans—The Future of Scientific Work in the Sudan—Sleeping Sickness in the Anglo-Egyptian Sudan—Investigation of Natural Conditions in the Bahr-El-Ghazal—Animal Trypanosomes—*Trypanosoma brucei*—Human Spirochaetosis—The Spirochaete of Egyptian Relapsing Fever—Spirochaetosis of Sudanese Fowls—Method of Obtaining Blood Aseptically for the Culture of Haematozoa in the Tropics—Fallacies and Puzzles of Blood Examination—Kala-azar in the Kassala and Blue Nile Districts and Eastern Sudan—General and Pathological Reports of the Kala-azar Commission—The Alkalinity of the Blood Serum in Kala-azar—*Herpetomonas lygaei*—Descriptions of Cases of Kala-azar, Non-Ulcerating Sore, "Oriental Sore," Parasitic Granuloma, Veldt Sore, *Ulcus tropicum*, Leucoderma—Fevers in the Sudan—Pyrexia—Diphtheria in the Tropics—Some Aspects of Tropical Sanitation—Sanitary Notes—The Water-supply of Towns in the Tropics—Lactose-fermenting bacilli in Surface Water, etc.—Filtering Properties of the Zeer—Human Botryomycosis—Veterinary Notes—Miscellaneous Notes on Haematozoa, Howell-Horrocks Bodies in the Human Blood, Mycetoma, Leprosy, Dysentery, Peculiar Bodies in the Intestinal Lymphoid Follicles of an Egyptian—Routine Work—Index.

The foregoing list of subjects of which this volume treats indicates the important character of the publication. The book is illustrated throughout in an excellent manner.

Contents of *Volume B*—General Science. Introduction—Report of the Chemical Section: A Test for Hashish, Khartoum Water-supply, Sobat River Water, Mechanical Analysis of Arid Soils, Soils of the Gezira, Gypsum as a Fertiliser for Sudan Soils, Gum Research, Experiments on Gum Production in Kordofan, Preliminary Notes on the Chemistry of the Latex of *Calotropis procera*—Report of the Entomological Section—Insects Injurious to Man and Animals—Animals Injurious to Farm and Garden Crops—Plant Diseases Ordinance—A New Genus and Two New Species of Culicidae from the Sudan—The Finches and Weaver Birds of the Sudan—Scorpions and Allied Annulated Spiders of the Anglo-Egyptian Sudan—Two Specimens of Spitting-Snakes from Southern Rhodesia—Sudan Spitting-Snake—*Herpetomonas aspongopi*—Ancient Gold Mining in the Sudan—Cult of Nyakang and the Divine Kings of the Shilluk—Some Tribal Customs in Their Relation to the Medicine and Morals of the Nyam-Nyam and Gour Peoples inhabiting the Bahr-El-Ghazal—Municipal Engineering Problems in the Tropics—Index.

The report of the Entomological Section by H. H. King is important to parasitologists, since it relates to Culicidae, Tabanidae (excellent coloured plates), and other blood-sucking and parasitic insects, ticks, etc. A new genus (*Diceromyia*) and two new species of mosquitoes are described by F. V. Theobald. The paper on *Herpetomonas aspongopi* is reprinted verbatim from *Parasitology* (1909), Vol. II, p. 202, unfortunately without acknowledgement, this doubtless owing to an oversight on the part of the Editor. N.

Looss, A. (1911). The Anatomy and Life History of *Agchylostoma duodenale* Dub. A Monograph. *Records of the School of Medicine*, Vol. IV, pp. 163–613, with plates XI–XIX. Cairo: National Printing Department (Ministry of Education), Egypt. 31 × 24 cm.

The compilation of this, the Second Part of Looss's classical monograph, has been awaited for a long time and is, perhaps for this reason, doubly welcome. Part II deals with the development of *A. duodenale* in the free state and constitutes a contribution of the first order which every parasitologist interested in the subject will necessarily be obliged to consult. Section I deals with the comparative anatomy, classification and development of Nematodes, with special consideration of those points which have led earlier authors astray. Section II deals in particular with *A. duodenale* and its development, the subject being treated exhaustively from every point of view. A full bibliography, Authors' and Subject Index, conclude the work which is illustrated by nine excellent plates.

REPRINTS.

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Hunter, W. D. and Bishopp, F. C. (1911). The Rocky Mountain Spotted Fever Tick. With special reference to the problem of its control in the Bitter Root Valley in Montana. *U.S. Dep't Agricult., Bur. Entomol., Bull. No.* 105, Washington. 47 pp., 3 pls., 3 figs.

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Terry, B. T. (1911). Chemo-therapeutic trypanosome studies with special reference to the immunity following cure. *Monographs Rockefeller Inst. Med. Res.* New York. No. 3, 33 pp.

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